Historic, archived document

Do not assume content reflects current scientific knowledge, policies, or practices.

LIBRARY
BUREAU OF
AUG 17 1938
AGRICULTURAL
ECONOMICS
U. S. DEPARTMENT OF AGRICULTURE

UNITED STATES DEPARTMENT OF AGRICULTURE

Miscellaneous Publication No. 292

Washington, D. C. Issued June 1938

20.1

MARKET DISEASES OF FRUITS AND VEGETABLES: CRUCIFERS AND CUCURBITS

By

GLEN B. RAMSEY
Senior Pathologist

JAMES S. WIANT
Associate Pathologist
Division of Fruit and Vegetable Crops and Diseases
Bureau of Plant Industry

and

GEORGE K. K. LINK
Professor of Botany, University of Chicago
and Collaborator, Bureau of Plant Industry

For sale by the Superintendent of Documents, Washington, D. C. - - - - - - - - - Price 45 cents

UNITED STATES DEPARTMENT OF AGRICULTURE

MISCELLANEOUS PUBLICATION No. 292

Washington, D. C. Issued June 1938

MARKET DISEASES OF FRUITS AND VEGETABLES: CRUCIFERS AND CUCURBITS[1]

By GLEN B. RAMSEY, *senior pathologist*, and JAMES S. WIANT, *associate pathologist, Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry*, and GEORGE K. K. LINK, *professor of botany, University of Chicago, and collaborator, Bureau of Plant Industry*

CONTENTS

	Page
Crucifers	2
Cabbage	2
Alternaria leaf spot	4
Bacterial soft rot	5
Black leaf speck	8
Blackleg	8
Black rot	10
Clubroot	12
Downy mildew	13
Edema	15
Freezing injury	15
Gray mold rot	17
Mosaic	17
Pythium rot	17
Rhizoctonia head rot	18
Rhizopus soft rot	20
Sunscald	20
Tipburn	20
Watery soft rot (sclerotinia rot)	21
Yellows	23
Chinese cabbage	24
Brussels sprouts	24
Cauliflower	25
Bacterial leaf spot	26
Bitter brown rot (browning)	26
Brown rot	27
Ring spot	28
Collards and kale	30
Cress	30
Horseradish	30
Bacterial root rot	31
Black root rot	31
Kohlrabi	31
Mustard	32
Radish	32
Black root	32
White rust	33
Rutabaga and turnip	34
Alternaria root rot and leaf spot	34
Anthracnose	35
Brown heart	35
White spot	36
Cucurbits	36
Cucumber	36
Anthracnose	37
Bacterial soft rot	39
Bacterial spot (angular leaf spot)	39
Cucurbits—Continued.	
Cucumber—Continued.	
Bacterial wilt	40
Cottony leak	41
Downy mildew	42
Fusarium rot	43
Low temperature break-down	43
Mosaic	43
Mycosphaerella black rot (mycosphaerella wilt; gummy stem blight)	44
Pink mold rot	45
Powdery mildew	45
Rhizopus soft rot	45
Scab	45
Watery soft rot	45
Muskmelon	46
Alternaria rot	46
Anthracnose	47
Bacterial soft rot	48
Blue mold rot	48
Charcoal rot	49
Cladosporium rot	49
Diplodia rot	50
Fusarium rot	51
Low temperature break-down	52
Macrosporium leaf blight	52
Phytophthora rot	53
Pink mold rot	54
Powdery mildew	54
Rhizopus soft rot	55
Pumpkins	56
Squash	56
Choanephora fruit rot (blossom rot)	57
Mycosphaerella black rot (mycosphaerella wilt; gummy stem blight)	57
Watermelon	58
Anthracnose	58
Chemical and mechanical injury	59
Fusarium wilt	60
Mycosphaerella black rot (mycosphaerella wilt; gummy stem blight)	60
Pythium rot and phytophthora rot	61
Sclerotium rot	62
Stem end rot	62
Literature cited	64
Crucifers	64
Cucurbits	68

[1] This publication is the fifth in a series designed to aid in the recognition and identification of pathological conditions of economic importance affecting fruits and vegetables in the channels of marketing, with a view to facilitating the market inspection of those food products and reducing and preventing losses from such conditions. The series represents an extended revision and elaboration, with the addition of illustrations, of a pre-

23735°—38——1

CRUCIFERS

The crucifers belong to what is known as the mustard family, which includes a large number of wild plants and the following food plants: Cabbage (*Brassica oleracea* L.), various types; Chinese cabbage or petsai (*B. pekinensis* (Lour.) Gagnep; rape (*B. napus* L.); turnip (*B. rapa* L.); Swede or rutabaga (*B. napo-brassica* DC.); pakchoi cabbage (*B. chinensis* L.); wild mustard (*B. alba* (L.) Boiss.); charlock (*B. arvensis* (L.) Kuntze); black mustard (*B. nigra* (L.) Koch); curled mustard (*B. juncea crispifolia* Bailey); radish (*Raphanus sativus* L.); garden cress (*Lepidium sativum* L.); horseradish (*Radicula armoracia* (L.) B. L. Robinson); and water cress (*R. nasturtium-aquaticum* (L.) Britten and Rendle).

CABBAGE

Cabbage is used for food as salad, as a cooked vegetable, and a very considerable amount is fermented and eaten as sauerkraut. Cabbage, like most other cultivated crucifers, belongs to the genus *Brassica*. The various members of the cabbage group supposedly are horticultural varieties which have been developed by cultivation from the wild *Brassica oleracea*. Thus the headless types (kale and collard, etc.) are *B. oleracea* var. *viridis* L.; the budding type (brussels sprouts) var. *gemmifera* DC.; the floral head types (cauliflower) var. *botrytis* L. and (flowering broccoli) var. *italica* Plenck; the enlarged stem type (kohlrabi) var. *gongylodes* L.; and the leafy head type (cabbage) var. *capitata* L.

The edible part of the cabbage plant consists of fleshy overlapping leaves forming a head, which are borne on a central stem with very short internodes. New leaves are formed by, and most active growth occurs in the region of, the terminal bud. The tendency toward formation of hard heads is much more pronounced in some strains than in others. The Danish type tends to produce harder heads than the domestic and pointed types. If growth is stopped by drought or other factors after the head is well-developed, and resumed subsequently, bursting and splitting result from the pressure generated within by the terminal bud. The same phenomenon occurs when the plant resumes growth the second season preliminary to developing its floral and seed stalk.

The outer, food-making leaves of the cabbage plant are green; the inner, shaded ones have the yellowish-white color characteristic of etiolated organs; however, the yellowish white of these leaves is of an entirely different cast from the yellow that is found in outer leaves

---

liminary publication entitled "Handbook of the Diseases of Vegetables Occurring Under Market, Storage, and Transit Conditions," by George K. K. Link and Max W. Gardner, which was published by the Department of Agriculture in 1919 especially for the use of the food-products inspectors of the Bureau of Agricultural Economics and was never distributed to the public. The material is organized on the basis of the botanical families to which the plants belong, but no botanical system is followed in arranging these families. Practical conditions make it desirable to issue the material in separate sections arranged somewhat in the order of the economic importance of the crops. The Host Index of the Fungi of North America, by A. B. Seymour, 1929, is used in the main as a guide to the nomenclature of causal fungi and to the names of authorities therefor. The colored plates, except as noted below, are reproduced from colored photographs prepared through the collaboration of Webster Bros., Chicago, Ill., under the direction of George K. K. Link, Max W. Gardner, and Glen B. Ramsey. The originals of plates 10, *B*, 14, and 18 were photographed in the Bureau of Plant Industry and colored by L. C. C. Krieger. Plates 3, *B*, and 12, *A* and *B*, are from photographs furnished by Charles Drechsler, and plate 8 is from photographs furnished by James L. Weimer.

when these become diseased or old, or when they are borne on sickly plants. In the purple cabbage the green of the outer leaves is somewhat masked and the purple of the innermost is lighter and frequently occurs only along the midrib. In the Savoy type the leaves are crinkly.

The marketability of cabbage for table purposes is affected by many factors, the foremost being crispness, solidity of head, color, and freedom from blemishes and decay. Since field diseases often affect the crispness, solidity, and color of cabbage, they are both a direct and an indirect factor in its marketability.

Lack of crispness may be due to poor growing conditions, to field diseases, to excessive drying out in storage, or to the respiratory activities of the tissues. Excessive loss of crispness because of respiration may result from too high a temperature during transit and storage or from too long a transit or storage period. Factors which determine solidity are strains, trueness to type, field diseases, and growing conditions, such as water and food supply in the soil. As pointed out before, growth of the cabbage head takes place from within. Anything that checks growth at the critical time is likely to lead to a loose, puffy head. Yellows, black rot, insect ravages, drought, or excessively hot weather, therefore, often lead to puffy, ill-shaped heads. At times puffiness is due to a resumption of growth in storage. High temperatures and high humidity may lead to activity of the terminal bud at the expense of the rest of the head. The growth made is slight, but this, together with the removal of food materials to the bud, may result in a soft, open head. Depletion of food materials, together with wilting and aging, at times cause the leaves to separate from the stem. If the leaves are tightly wrapped, they may remain part of the head; if not, they fall off, leaving the leaf scars, which show the traces of the vascular bundles and bear small to well-developed buds above them. Certain field diseases (yellows, black rot, and blackleg) frequently lead to this phenomenon.

The color requirements for early and late or storage cabbage are somewhat different. Early or freshly harvested cabbage, especially of the pointed or Savoy type, should have healthy green leaves. Yellowing of leaves is an undesirable feature that may be due to disease or to break-down from respiration in transit or storage. The inner, protected leaves of early cabbage and of good storage cabbage have a yellowish-white color quite different from the yellow found in leaves that previously had an abundance of chlorophyll. When cabbage gets old or is exposed to conditions that cause rapid respiration, even the blanched leaves may take on a sickly yellow color.

Several of the most serious diseases of cabbage affect its marketability only indirectly by causing small, puffy, ill-shaped, poorly colored, or bitter heads. Blackleg (*Phoma lingam* (Tode) Desm.), clubroot (*Plasmodiophora brassicae* Wor.), malnutrition, and yellows (*Fusarium conglutinans* Wr.) are among the most serious maladies of cabbage in the field, yet they do not cause any lesions of the head itself.

Other cabbage diseases occurring in the field but of minor importance so far as marketing is concerned are anthracnose (*Colletotrichum higginsianum* Sacc.), powdery mildew (*Erysiphe polygoni* DC.),

ring spot (*Mycosphaerella brassicicola* (Fr.) Lindau, p. 28), root knot (*Heterodera marioni* (Cornu.) Goodey), sclerotium rot (*Sclerotium rolfsii* Sacc.), and white rust (*Albugo candida* (Pers.) O. Kuntze, p. 33).

Several diseases which affect production seriously may also directly affect the marketability of the heads. Foremost among these is black rot (*Bacterium campestre* (Pam.) E. F. Smith). Others are alternaria leaf spot (*Alternaria brassicae* (Berk.) Sacc., *A. herculea* (Ell. and Mart.) J. A. Elliott and *A. oleracea* Milbrath), bacterial leaf spot (*Bacterium maculicola* (McC.) McCulloch); downy mildew (*Peronospora parasitica* (Pers.) DBy.), and head rot (*Rhizoctonia solani* Kühn).

There are several diseases which only occasionally cause losses in the field, but which are exceedingly important factors in the later stages of distribution and in storage. These are bacterial soft rot (*Bacillus* spp.), gray mold rot (*Botrytis* spp.), rhizopus soft rot (*Rhizopus nigricanus* Ehr.), and watery soft rot (*Sclerotinia sclerotiorum* (Lib.) DBy. Often these also cause heavy indirect losses in transit and storage by necessitating heavy trimming (*13, p. 49; 21, p. 378; 28; 36; 63; 65; 71; 74; 79; 87; 158*).[2]

ALTERNARIA LEAF SPOT

(*Alternaria brassicae* (Berk.) Sacc., *A. herculea* (Ell. and Mart.) J. A. Elliott and *A. oleracea* Milbrath)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Alternaria leaf spot, sometimes called black leaf spot, occurs on cabbage, collards, brussels sprouts, cauliflower, Chinese cabbage, and rutabaga. Although it occurs in the field where the primary infection takes place, it is principally a transit and storage disease. On cabbage this disease is restricted to the leaves; on cauliflower it sometimes affects the curd. The market losses are due to decay which the fungus causes, to secondary decay for which it opens the way, and to blemishes which render heads unsightly and necessitate heavy trimming.

Alternaria leaf spot caused by *Alternaria brassicae* is widely distributed. *A. herculea* and *A. oleracea* have also been reported to cause black mold rot (pl. 1, *D*) in the region around San Francisco, Calif. The alternaria leaf diseases may become serious in storage houses if temperature and humidity are not well controlled.

The first symptom of the disease is the appearance of small, grayish-brown to black specks less than one-sixteenth of an inch in diameter (pl. 1, *A*). They may appear at any time during the life of the cabbage head, but usually develop first and most abundantly on the outer, older leaves of plants in the field or in storage (pl. 1, *B*, *C*, *D*). Often countless specks appear simultaneously on the leaf blades. As the spots enlarge they lose some of their circular shape and become sunken and black, sometimes showing a purplish cast with the center darker than the margin. The color of the spots depends somewhat on the color of the leaf affected. On pale-green leaves of the Winningstadt and Cannon Ball varieties, or on cab-

[2] Italic numbers in parentheses refer to Literature Cited, p. 64.

A, *B*, *C*, Alternaria leaf spot on outer green leaves of cabbage; *D*, alternaria leaf spot and black mold rot in storage cabbage.

bage that has been stored for a while, the spots are lighter than on the dark-green varieties that have not been stored. The spots vary in size from one-twentieth of an inch to 1 inch or more in diameter. They may remain single but frequently coalesce, especially in storage. The type of spot due to *A. brassicae* is marked by concentric brown zones and frequently by a growth of brown to black mold. The spots caused by *A. oleracea* are not zonate, and usually are not covered by a moldy growth until the leaves turn yellow or are shed. Those caused by *A. herculea* are usually smaller and grayer in color than those produced by the other organisms. Under dry conditions, the necrotic areas generally fall out, leaving holes; under moist storage conditions the underlying tissues become involved and often severe decay results (pl. 1, *D*). In many instances secondary organisms, such as bacteria, follow in these lesions.

CAUSAL FACTORS

Three causal organisms have been reported for this type of disease—*Alternaria brassicae*, which is generally thought to be responsible for most of the losses, and *A. oleracea* Milbrath and *A. herculea* (Ell. and Mart.) J. A. Elliott, which are reported from California. *A. herculea* has been found in other States but apparently is seldom of economic importance. High humidity favors the spread and development of this disease. Low temperatures (35° to 45° F.) and low humidity greatly inhibit the development of decay.

CONTROL MEASURES

The following precautions are recommended as possible control measures: (1) Disinfection of the storage house by spraying the walls, benches, and bins with 4–4–50 bordeaux mixture, (2) care in handling so as to minimize injury to the heads, (3) maintenance of a storage temperature of 33° to 34° F., and (4) keeping the humidity as low as possible by proper ventilation. Affected stock can be trimmed and put into salable condition (*52*, *74*, *79*, *82*).

BACTERIAL SOFT ROT

(*Bacillus carotovorus* L. R. Jones)

OCCURRENCE, SYMPTOMS, AND EFFECTS

The soft rot of cabbage probably causes more losses on the market than any other cabbage disease. This is due to the fact that bruised or wounded tissues are readily affected, and that this causal organism is a secondary invader of tissues killed or weakened by most of the parasitic and some nonparasitic diseases of cabbage. Thus it generally follows yellows, black rot, and clubroot and is responsible for most of the damage following freezing.

Ordinarily, soft rot is of no significance in the field. However, under very favorable conditions it is known to attack the pointed tip of the heads of Winningstadt, Wakefield, and similar varieties.

The symptoms vary somewhat in leafy and in fleshy organs. The outstanding ones are a soft, mushy, sticky decay and a very offensive odor. In the cabbage, as in other fleshy tissues such as carrot, the colorless or yellow tissues are only slightly discolored at first; in

green tissues the water-soaked areas have a muddy green or greasy appearance, finally turning slightly brown (pl. 2, *A*). The first symptom of the rot is a water-soaked area with a rather definite border. This enlarges rapidly if the temperature is high and the air moist. In moist air it continues wet until all the tissues are destroyed; in dry air it dries out rapidly, leaving a brittle leaf mass or a cavity in the stem.

The pith of stems or roots is very often affected, and the resultant condition in cabbage is often referred to as "stump rot." Stump rot usually follows infection during cutting or following freezing. Unless care is taken in cutting to avoid heads affected by the bacterial rot, it is easy to carry bacteria on the knife from diseased heads to the freshly cut stems, which provide ideal starting places for decay if they are moist. In root crops, such as carrot, turnip, and parsnip, the lesions usually occur in the pith, starting in at the crown of the plant. In cabbage the lesions usually are on the surface (pl. 2, *A*) or in the pith, following bruises or wounds. In the former case the outer decayed tissues can be removed and the progress of the disease arrested by drying the freshly exposed surface. If the stem is invaded or the disease penetrates deep into the head following black rot, the entire head is worthless.

CAUSAL FACTORS

The complex of diseases grouped as bacterial soft rots is caused by bacteria of the *Bacillus carotovorus* group. *B. carotovorus* attacks a variety of fleshy vegetables, including cabbage, carrot, celery, cucumber, eggplant, muskmelon, onion, parsnip, pepper, potato, radish, rutabaga, tomato, and turnip. A related species, *B. melonis* Gidd. (*B. aroideae* Town.), causes a similar rot. Another related form is *B. apiovorus* H. Wormald, which attacks celery, the calla bulb, cucumber, and iris. In view of the fact that the rots caused by these organisms behave so similarly, the data determined for the rot caused by *B. carotovorus* will be applied to them as though they were a unit.

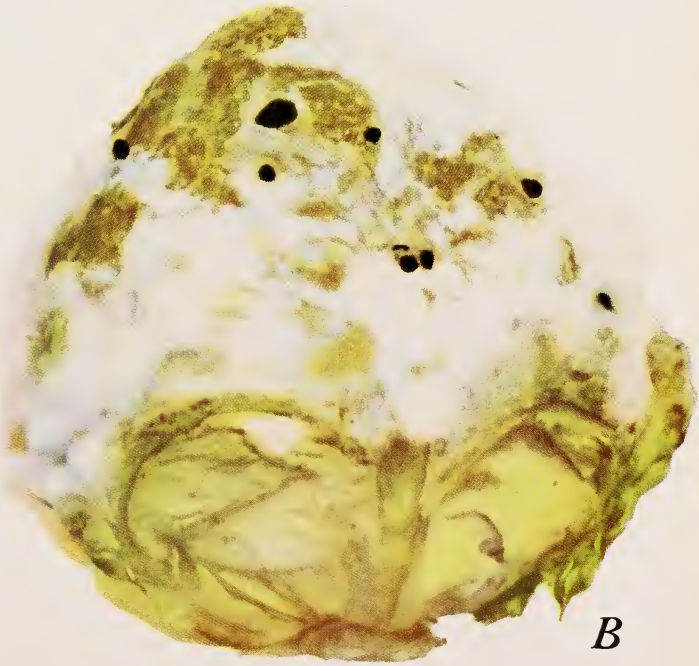
Practically all invasion of tissue by the soft rot organism is through wounds, the lesions of other diseases, or tissues weakened by mechanical agencies, freezing, asphyxiation, or aging. The organisms will only attack injured or dead tissues, and these only when they are wet. Breaks in the tissues or moistened surfaces provide the water and food they need to get started and to carry on growth and reproduction.

Soft rot may be spread in transit and storage by contact, or by the oozing or dripping of the decayed tissues. There probably is much less actual spread of this decay in transit than of many fungus rots, because there is no mycelium.

The organism is particularly sensitive to desiccation and to direct sunlight.

Wet weather favors the disease in the field directly and indirectly. It brings about a multiplication of the organism, disseminates it, moistens plant tissues, and makes them more turgid, or suffuses them with water so that they break or crack.

Warm weather in general favors the rot, but temperatures between 69° and 77° F. are most favorable because there is less drying



A, Bacterial soft rot of cabbage; *B*, sclerotinia rot of cabbage.

out of tissues. At 91° the organism does not grow as well as at 69°; at 61° it produces only one-half and at 54° only one-third as much rot as at 69°, while at 46° there is practically no invasion of tissues.

CONTROL MEASURES

As with many other storage rots, the control of bacterial soft rot begins in the field. The first step is the prevention of other parasitic field diseases in the crop that increase the reservoir of inoculum. Plants killed by yellows frequently become affected with soft rot and serve as sources of infection in the field.

Crop rotation will not only aid in the control of certain field diseases but will also minimize the reservoir of soft-rot bacteria in the soil. The danger of contamination in field and packing house may also be greatly reduced by a reasonable degree of sanitation in the disposal of culls, trash, and litter. Every precaution should be taken to avoid unnecessary contamination of vegetables during harvesting and packing. Rotting cabbage and cauliflower heads should not be cut when the crop is harvested because the knife may carry infection to the freshly cut stumps of sound heads. If root crops must be washed, the water should be changed frequently. Rotting radishes, rutabagas, and turnips should be sorted out and disposed of before storage, and no rotting vegetables should be thrown onto manure or compost heaps to be eventually returned to the field. In fact, every precaution should be exercised to guard against the use of contaminated manure or compost.

Proper handling during harvest to avoid all unnecessary wounds, cuts, bruises, sunscald, and freezing injury is of primary importance in soft rot prevention. Furthermore, many root crops may profitably be allowed to dry off before storing or shipping. This will kill the soft rot bacteria on their surfaces and allow all surface wounds to become dry and resistant to infection.

Low temperature during transit and storage is of course the most important factor in the control of this rot. Ventilation is also important, because by reducing the relative humidity and preventing the condensation of moisture on surfaces, it keeps the tissues less turgid and therefore less susceptible to infection. Furthermore, it disposes of the products of respiration, including heat, and prevents asphyxiation injury, which predisposes vegetables to soft-rot infection. Storage pits, bins, and houses should be properly constructed so as to provide for the necessary ventilation. Where refrigeration is impossible, much can be accomplished by maintaining good ventilation and as low temperatures as possible for vegetables while in transit and storage.

Temperature control during storage and transit is probably relied upon to a greater extent than any other factor for the prevention of soft rot. Soft rot will not make much progress in refrigerator cars after the products have once become cooled.

Because of the offensiveness of the bacterial soft rot, salvaging and reconditioning partially affected lots are less feasible than with the fungus rots. However, by careful sorting or trimming, badly damaged shipments may be salvaged, provided the stock can be used at once, or exposed to dry air, especially in direct sunlight (*2; 4; 28; 36; 37; 65; 66, pp. 300–313; 71; 79*).

BLACK LEAF SPECK

Black leaf speck is characterized by small, sharply sunken, brown or black specks occurring anywhere on the leaves of cabbage, cauliflower, and Chinese cabbage. At times they are found in great abundance, especially on the leaf blades. These specks may occur in a few of the outer head leaves only, or in leaves throughout the head (pl. 3, *A*). Frequently it is difficult to distinguish them from the early stages of alternaria spot, which occurs in California cabbage, and from ring spot as it appears in Oregon cauliflower. The spots render these products unsightly, thus affecting their marketability. They also tend to lower their resistance to storage rots.

Black leaf speck may occur in cabbage, Chinese cabbage, and cauliflower from all sections. Very little is known about the factors that bring it about, save that it is not caused by organisms. It occurs commonly in transit and storage and for this reason might be classed as a break-down associated with low temperature. It also occurs sometimes in the field, where it has been found, in some instances at least, to have been definitely associated with a decided drop in temperature.

Black leaf speck has been produced experimentally by placing cabbage at room temperature in an atmosphere having little or no oxygen; and also by holding it in a well-ventilated room at temperatures near 32° F. for 2 or 3 weeks. Control apparently involves providing good ventilation in transit and storage, and maintaining temperatures between 35° and 40° F. (*54*).

BLACKLEG

(*Phoma lingam* (Tode) Desm.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Blackleg occurs on cabbage, cauliflower, turnip, rutabaga, rape, brussels sprouts, kohlrabi, kale, and white and black mustard.

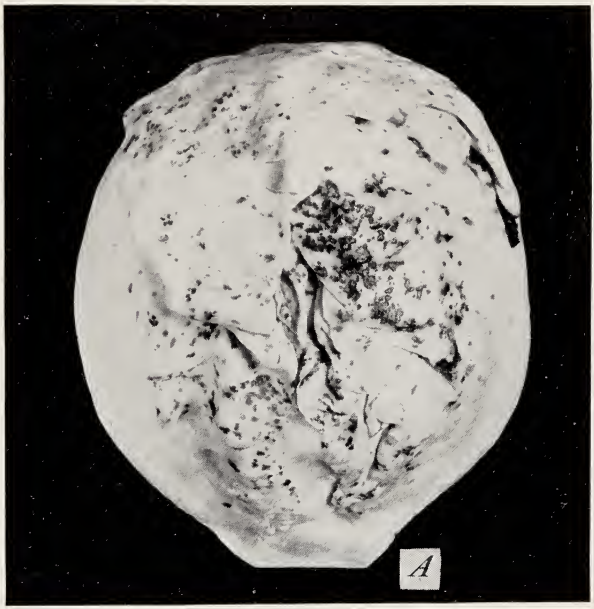
The disease may affect the cabbage plants throughout their entire life, infection taking place in the seedling stage as well as later on. All parts of the plants are susceptible. The fungus destroys primarily the parenchyma, and thus induces secondary systemic symptoms, when the absorbing and conducting tissues of the roots and stem are severely involved.

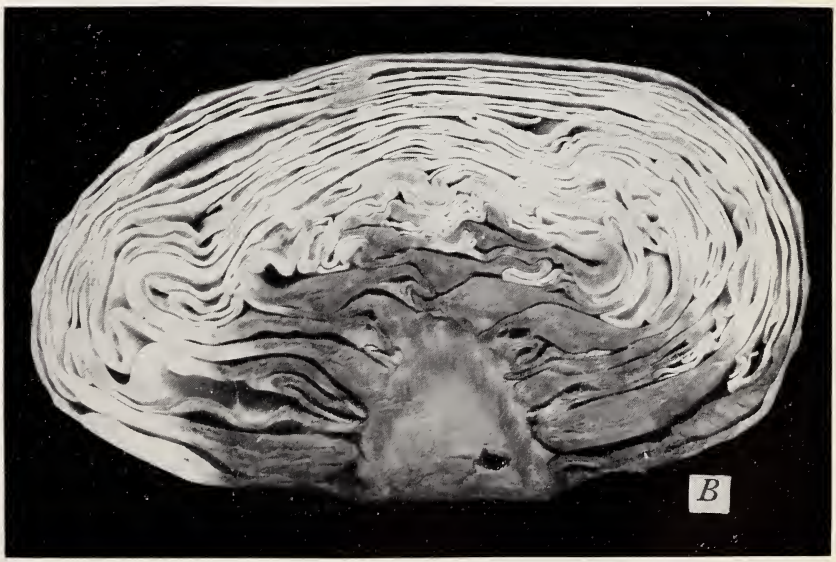
Blackleg is of great importance in the field because of the destruction of plants. From a market point of view, it is only of indirect importance because of its responsibility for poorly shaped, poorly formed, and small-sized heads.

The disease occurs very widely in the United States, England, the Netherlands, France, Germany, and Australia. To a very great extent its occurrence and severity are determined by climatic factors, especially rainfall.

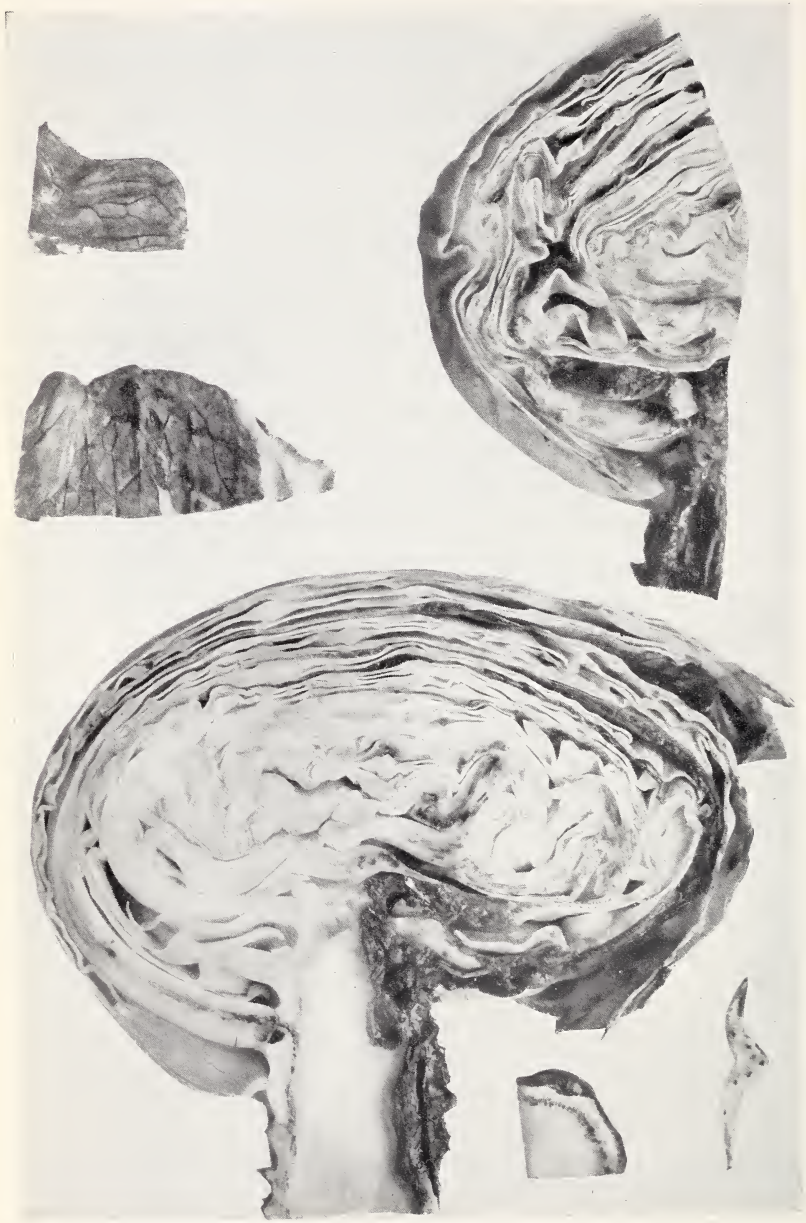
In Europe and New Zealand a serious dry rot of swedes and turnips in storage is attributed to the blackleg fungus, or one very closely related to it.

The disease may occur in cabbage plants in all stages of development. If the cotyledons are affected they usually wither and die. The stems of seedlings collapse and become grayish white in color. Later the lesions bear the pycnidia (fruit bodies) of the fungus.





A, Black leaf speck of cabbage; *B*, pythium rot of cabbage.



Black rot of cabbage.

The pycnidia are light at first but finally turn brown. In them are borne innumerable spores of the fungus, which upon attaining maturity are exuded during rains or humid weather. On the older leaves, stems, and pods, the lesions usually have purplish borders and grayish-white centers bearing an abundance of pycnidia. Often the stems are girdled below or at the ground level by lesions which, because of their brownish-black color, have given rise to the name "black-leg." The fibrous root system is often reduced or entirely destroyed. Destruction of the roots and girdling of the stem lead to the curtailment of the water supply and to the interruption of the movement of food into the roots. Lesions on the leaves interfere with the food-making functions, as a result of which the plants are stunted and, in severe cases, they wilt and die. Often purpling of the leaves precedes wilting.

Severely infected seeds are shriveled and dead and may contain an abundance of pycnidia. Less severely infected seeds are not always killed and give no external evidence of being diseased.

CAUSAL FACTORS

The causal fungus is *Phoma lingam*. It overwinters on seed heads in storage, in infected seed, and on infected stems and refuse in the field. In the field primary infection of seedlings occurs 10 to 15 days after planting. The lesions develop pycnidia full of spores, which because of rains or dew are discharged from the pycnidia and then are spattered by subsequent rains from the lesions or the soil to new sites of infection. Later, infected seed plants are carried into the field and serve as foci of infection. Moisture is a much more important limiting factor than temperature. Under greenhouse conditions, with a temperature of 70° to 75° F. and relative humidity of 60 to 70 percent, the first signs of the disease appear in 8 to 10 days; other primary lesions continue to develop for 3 to 5 weeks. Generally the first symptoms appear on the cotyledons; hypocotyl infection comes later.

Sometimes the disease is a factor in storage. Plants that appear to be free from infection when put into storage may show small, dark spots in the vascular ring. If the storerooms become warm and moist, the disease may become serious by spring, progressing from the stem into the leaf, and attacking the outer leaves first. Trimming often is resorted to in order to keep the stock free from secondary decays and to give it an attractive appearance.

CONTROL MEASURES

Control of the disease involves the following measures: Destruction of diseased plants, the use of clean soil for the seedbed, selection of clean heads for seed production, and treatment of the seed by soaking for 30 minutes in a 1 to 1,000 solution of mercuric chloride. This kills surface contamination of the seed but does not always kill the fungus in infected seeds, and such seeds unfortunately may not show any external evidence of being diseased. Treatment of the seeds for 30 minutes with water at a temperature of 122° F., without previous soaking, gives complete disinfection with some reduction in germination. Covering the seedbed with cheesecloth to prevent rain spattering has been found helpful in reducing disease. Growing cabbage

seed in sections in which there is practically no rainfall after planting (Puget Sound section) has proved to be an effective procedure (*15*, *30*, *33*, *50*, *69*, *70*, *71*, *73*).

BLACK ROT

(*Bacterium campestre* (Pam) E. F. Smith)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Black rot is found in practically all the cultivated species and varieties, as well as some wild species, of the crucifers. The cabbage plant is susceptible practically throughout its entire life, because the seedlings as well as the maturing plants are infected. The vascular elements are the primary place of entry and progress for the bacteria. The adjoining tissues are involved secondarily. The disease occurs primarily in the field, where the damage caused may range from slight to total loss of the crop. It is also a direct and an indirect cause of losses in transit and storage. From the point of view of production, the losses are due to killing of seedlings, to increase in the number of heads of small size, poor shape, and with discolored tissues, and frequently to secondary rots in heads produced by diseased plants. On the market, losses are due to vascular and parenchyma discolorations and to secondary bacterial soft rot. At times, when the head is not affected throughout, some salvage can be effected by trimming off blemished or decayed leaves.

Black rot is a vascular disease. It is not a soft rot, although it may be complicated by soft rots. The most pronounced symptom is a black stain in the vascular system of the stem and leaves accompanied by a yellowing of the foliage. Often definite patches of yellow or light brown, with a sharply marked network of black veins, appear in the leaves. In case of marginal invasion of the leaves, affected areas are more or less wedge-shaped, with the apex of the wedge extending into the leaf toward the midrib.

When the plant is badly diseased there is a gradual or successive shedding of affected leaves, so that often only a small tuft of leaves remains at the end of a long bare stalk. At times there seems to be a stimulated development of buds in the axils of shed leaves. When such a stem is cut the characteristic blackening is found in the vascular ring (pl. 4). The discoloration may involve the entire ring, but more often only one side or isolated bundles. By sectioning, one can establish the connection between the stem discoloration and the staining in the leaves. The stain may not be visible externally in thick midribs, but it shows through as grayish-black lines in thinner ones. Generally, the cortex and pith are not involved. Unless the base of the stem and the roots are infected before they become woody, they remain free from bacteria and stain. Discoloration found in the woody part generally indicates that the plant became infected in the seedling stage. Usually when cabbage plants are attacked in that stage or a little later, they are either killed outright or are so injured that they do not form heads. Severe seedling infection is characterized by blackening of the cotyledons, by sudden wilting, and by death.

Although black rot of cauliflower usually develops as in cabbage it sometimes manifests quite different symptoms. In Long Island it

is reported that rains may so favor the development of the initial lesions that halves of leaves may quickly wilt without showing the usual black discolorations. Infection of the lower leaves also occurs through the under surfaces where water drops form and hold the bacteria. In such cases large water-soaked lesions are produced, and the leaves soon wilt and dry, thus giving the plants a blighted appearance.

In kohlrabi affected with black rot the staining of the vascular elements often is very pronounced in the enlarged stem without any externally visible symptoms, and cavities frequently occur. In Chinese cabbage numerous small brown spots may occur on the leaf blades along with vascular discolorations. In turnips this disease sometimes causes a dwarfing of the roots and the formation of cavities which may not be visible externally. In none of these products is there a definite odor associated with the black-rot lesions unless some secondary organism has contaminated the affected tissues.

CAUSAL FACTORS

The causal agent, *Bacterium campestre*, may overwinter on seed, in or on mother cabbage heads, in plant trash, or, in favorable sections, in the soil. Primary infection of the cotyledons occurs in the seedbed 12 to 20 days after planting the seed. From infected seedlings and mother heads, or from the soil, the bacterium is disseminated by splashing water, by insects, and by man in the course of cultural operations.

Infection does not seem to take place through the roots; it can take place through wounds.

Most infections of cabbage in the field appear to take place through the water pores in the margin of the leaves. Droplets of water are exuded at night and may remain over the pores for several hours after sunrise during moist, cloudy weather. If black-rot bacteria get into these drops, they may begin growth and work their way into the substomatal chamber. In 6 to 10 days there may be a slight darkening of the infected leaf tooth; generally 11 to 20 days elapse before the bacteria get into the veins of the leaf. Progress into the leaf from water pores is usually by way of the spiral vessels; entrance into the stem is effected through the leaf traces. The pathogen spreads up and down in the stem if the tissues are soft, frequently entering new leaves from the stem. If the leaves are severely affected from the stem, they may wilt, become unjointed, and fall.

Temperature, moisture, air, and soil are factors which govern infection. The lowest temperature at which the organism can grow is about 40° F.; the highest 100° to 102°; it grows best at about 86°. Consequently, the time of appearance and severity of the disease are very largely determined by weather conditions. So far as growth of the organism is concerned, hot weather is favorable. For infection, however, warm and rainy or moist weather with cool nights is more favorable than hot, dry weather. Cool, moist soils are more favorable for the disease than dry ones.

Black rot does not spread or develop in transit or in storage. However, it opens the way for serious secondary rots, especially bacterial soft rot, thus leading often to very heavy losses.

CONTROL MEASURES

Control of this disease involves sanitation, crop rotation, and seed and soil treatment.

Diseased plants or manure containing them should be kept off clean land. Great care should be exercised in selecting a seedbed. Soil that has not borne cruciferous plants or that at least has not borne any crop infected with black rot should be used.

Only heads free from black rot should be used for seed production. Seed should be treated with a mercuric chloride solution (1:1,000) for 30 minutes and then rinsed in water. If the disease appears in the seedbed, the whole lot should be discarded. The treatment of seed is of paramount importance, because it has been demonstrated that seedbed infection, which is responsible for the most serious losses, is brought about by planting contaminated seed.

It has been reported that treatment of cauliflower seed with 1:1,000 mercuric chloride solution for 30 minutes, or soaking with hot water (122° F.) for 25 to 30 minutes, or three separate treatments of the seedbed with the mercuric chloride solution, as for maggots, give fairly good control of black rot.

Cabbage seed grown in dry areas is usually free from black rot (*5; 10; 18; 27; 48; 56; 65, v. 2, pp. 300–334; 73; 77; 79*).

CLUBROOT

(*Plasmodiophora brassicae* Wor.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Clubroot occurs on nearly all cultivated crucifers as well as on many wild species of this family. The malformations that characterize the disease are found only in the parts below ground, with indirect secondary systemic symptoms.

Clubroot is exclusively a field disease. It is a direct factor in marketing only in root crops, such as turnip, rutabaga, and radish.

The outstanding symptom of clubroot is enlargement of the main or lateral roots or both. In cabbage the enlargements generally are spindle-shaped and occasionally lobed. In turnip and other crucifers the clubs are frequently globular; at times they assume enormous proportions, the diameter of a club often being 10 times that of a normal root. At first the clubs are dirty gray or pale yellow, smooth, firm, and even brittle; later they become dark colored, deeply cracked and furrowed, soft, and flabby. Finally, decomposition, due to secondary rots that start in the broken surfaces, changes the club into a semiliquid, dark-brown, foul-smelling mass.

CAUSAL FACTORS

The causal organism is *Plasmodiophora brassicae*. It hibernates in the debris of affected crops or in weeds of the previous season. The pathogen can be carried into a field either in the spore or amoeba form by manure or compost derived from infected plants. It can be carried by drainage or irrigation water, by wind-blown soil, and by seedlings infected in the seedbed. The organism may enter the root through the root hairs or through wounds.

Infection and severity of the disease are determined largely by temperature and soil moisture. The pathogen causes disease through a temperature range of 53.6° to 80.6° F., with most malignant development occurring at about 77° F. Most severe club formation occurs at the optimum water content for cabbage growth. There is very little infection if the soil has a moisture content less than 50 percent of the water-holding capacity, even though enough water may be available for cabbage development. In dry soils there may be infection of the roots without severe checking of the growth of the host. As the water content of the soil increases, severity of infection increases up to the saturation point of the soil. In wet soils the enlarged roots are frequently destroyed by secondary organisms, which cause foul odors and the death of the plant.

The pathogen is sensitive to alkalinity and consequently soils rich in lime usually are free from the disease, although some soils naturally high in lime have been found infested with the clubroot organism. The character of the soil also affects the severity of the disease on different hosts. Turnips are said to be more susceptible on sandy soils; cabbage, on heavy clay soils. Compacted soil is more favorable for the disease than friable, well-aerated, and well-drained soil.

CONTROL MEASURES

It has been reported (*85*) that treatment of infested soil with 1:1,500 mercuric chloride prevents infection of young plants through the critical period. Disinfection of the seedbed with steam or formaldehyde also has been found useful.

Fields can be kept clean by planting only seedlings grown in seedbeds free from clubroot, and by not using manure originating from the feeding of uncooked diseased plants. A 7-year rotation in which cruciferous plants are not included will rid the infested soil of the organism.

The use of lime is beneficial, but it is not always practicable. Two tons of hydrated lime per acre has given control in some heavily infected soils.

Resistant varieties of cabbage have not been developed satisfactorily, but turnips such as Snowball, Purple Top Milan, and White Egg are resistant, and the American Purple Top, Wilhelmsburger, and Bangholm varieties of rutabaga are highly resistant (*6*, *7*, *14*, *41*, *44*, *45*, *46*, *53*, *71*, *83*, *85*).

DOWNY MILDEW

(*Peronospora parasitica* (P.) Fr.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Practically all plants belonging to the mustard family are subject to attacks of the downy mildew fungus. The short, fuzzy, or downy mycelium on the surface of affected plants has given rise to the common name "downy mildew." Plants are more likely to suffer damage from this disease when they are in the seedbed. The disease is of direct importance, from the market point of view, only in stored cabbage and turnips and in those members of the mustard family whose tops are used as greens.

When seedlings of crucifers are attacked, small, pale-yellow, angular areas on the leaves are the first noticeable symptoms. A little later, the fine, downy mycelium and spores of the fungus become evident on the underside of the leaves. Badly affected leaves curl up and become dry. Occasionally the seedlings of plants such as cabbage are attacked so severely in the seedbed that the whole bed is ruined.

Although downy mildew is chiefly known as a seedling disease, it sometimes causes damage to the plants in the field during excessively humid seasons or prolonged rainy weather. On market stock it is comparatively unknown. In recent years, however, certain lots of stored cabbage have been found seriously affected with the downy mildew fungus. The external evidence of this trouble is a dark-gray to black discoloration of the outer leaves and pith region of the stump (pl. 5, *A*, *B*, *D*). On cutting the heads lengthwise it may be found that the discoloration extends throughout the length of the stalk (pl. 5, *A*). The advancing edge of the lesions produced are yellowish brown and the older portions of the lesions are grayish black. From the discolored leaf scars it is evident that the fungus entered the stalk from the infected lower leaves of full-grown cabbage. During storage the fungus works up through the pith and gradually spreads out through the parenchyma cells between the vascular bundles and into the inner head leaves. By contact the fungus passes from one inner leaf to another so that eventually a group of overlapping leaves become affected and turn grayish black. In some heads the minute terminal bud leaves have been found completely blackened (pl. 5, *C*.) No particular odor is produced in the discolored tissues, and there is no soft rot produced unless the affected tissues are invaded by bacteria. Alternaria rot and rhizopus soft rot sometimes follow in the tissues affected by downy mildew.

In storage turnips, the fungus has been observed to cause a brown and black discoloration of the root, extending from the crown down into the central cylinder. Affected tissues are firm and turgid; freshly invaded tissues are mottled or streaked and are light brown in color.

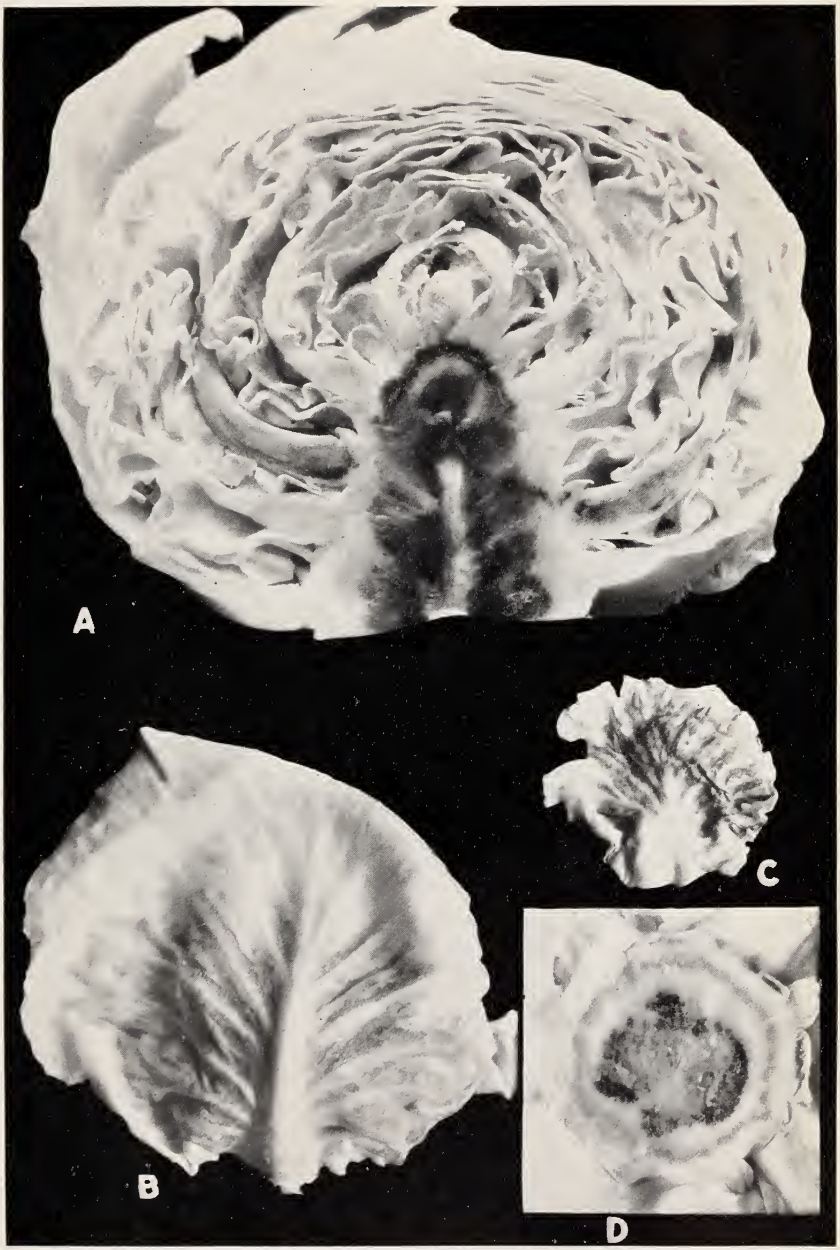
CAUSAL FACTORS

The fungus, *Peronospora parasitica*, overwinters in the form of resting spores (oospores) in debris of plants affected the previous year, or as a perennial mycelium in biennial or perennial host tissues. Either of these eventually gives rise to spores (conidia), which are disseminated by the wind or by water and which upon germination may cause new lesions on susceptible plants. Like other downy mildews, this fungus requires an abundance of moisture for spore production and spore germination.

The mycelium in turn gives rise to more conidia which spread the disease, and toward the end of the life of the host it produces resting spores. As just stated, the mycelium itself may live through the winter in fleshy tissues. The spores of this fungus germinate very readily in water at low temperatures (46° to 54° F.). At a temperature of 66° the spores do not germinate so well, and at 73° to 77° very few germinate.

CONTROL MEASURES

Ordinarily this disease is not of sufficient severity to make control measures necessary. It is advisable to destroy the weeds belonging to



A, Section through a cabbage head showing discoloration induced by *Peronospora*; *B* and *C*, inner head and heart leaves discolored by the downy mildew fungus; *D*, section across the stem at the base of a cabbage head showing discoloration of the pith region.

the mustard family, which might harbor the fungus, and sometimes it is well to rotate the crops or at least to change the location of the seedbeds. Seedbeds should not be sown too thickly nor be allowed to become excessively moist.

The fact that viable mycelium of Peronospora has been found in turnip roots and cabbage heads in the winter indicates that the disease may live over from year to year in turnips and cabbages that remain in the field. If possible, diseased produce should be removed from the field and destroyed (*24*, *61*, *71*, *79*).

EDEMA

OCCURRENCE, SYMPTOMS, AND EFFECTS

Frequently excrescences appear on the lower side of leaves of cabbage, cauliflower, tomatoes, and other crops. Only in such crops as cabbage, in which the leafy parts are marketed, do these overgrowths ever become a factor from the market point of view. Even in cabbage, edema is not a serious factor, because the badly affected leaves can be trimmed off. The lesions may blemish the leaves, but do not affect their keeping quality.

The lesions consist of small, wartlike outgrowths on the lower sides of the leaves. Sometimes there are corresponding depressions on the upper sides. The swellings may be far apart or closely crowded. In the early stages, the epidermis remains intact and the swelling is smooth; later, the epidermis is ruptured and the underlying cells are exposed as white, waxy appearing masses. Eventually they lose their spongy texture, becoming corky and yellow or brown.

The lesions are due to excessive development or growth of cells. This may be caused by (1) loss of balance between intake and outgo of water, so that the tissues become suffused with water; (2) copper sprays (ammoniacal copper carbonate) of such concentration that they injure the cells without killing them; and (3) mechanical injuries. In the greenhouse, the first cause usually is responsible. There the disease can be controlled by taking precautions to prevent the soil from becoming too warm or too wet and the air from becoming saturated with water (*1*, *86*).

FREEZING INJURY

It is a well-known fact that heavy losses of cabbage occur annually as a result of freezing, both in the field and after harvest. Cabbage tissue has one of the highest freezing points (31° F.) found among vegetables. Although cabbage tissues differ from those of many other vegetables, in that they can tolerate formation of ice and thawing without apparent injury, there is a limit below which they are likely to be injured. Sometimes frozen cabbage will thaw out without injury and sometimes it will not. In the former case the tissues are merely frozen, while in the latter they are frozen to death. However, there is no way of telling by examination of a frozen head of cabbage, or of other vegetables which act similarly, whether they will show injury and break-down when they thaw out. Immediately after thawing, the frozen areas appear water-soaked, due to suffusion with

water liberated by the thawing ice in the intercellular spaces. If the cells are still alive, some of the water will go back into them and the only effect in the tissue is a slight wilting or shriveling due to the excessive loss of water by transpiration and evaporation. Tissues responding in this way become flaccid, pithy, or spongy and tough, and lose their characteristic flavor. Some of them can withstand the freezing process several times before the effect becomes pronounced; others develop it immediately. If the cells are not alive, the water does not reenter them and is either lost rapidly into the air, with attendant drying out of the tissues, or it remains and the tissues become a leaking, disorganized mass, which is attacked by saprophytic fungi and bacteria.

Whether or not ice formation in the tissues is attended by permanent injury and death seems to depend upon the degree to which the temperature falls, the rate at which it falls, the duration of the critical temperature, and the condition of the plant tissues themselves. Some contend that a rapid rate of thawing leads to injury, and that a slow one does not.

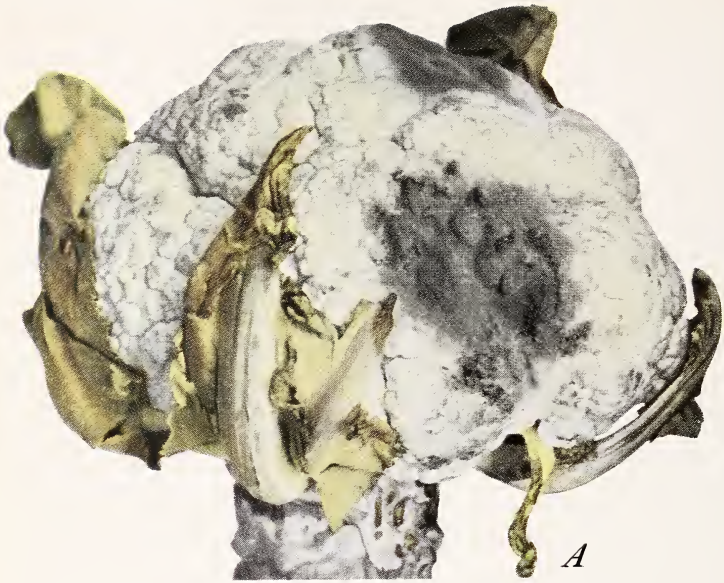
The presence of ice in cabbage tissues renders them rigid or brittle. Leafy tissues and even such storage tissues as turnips and rutabagas lose their natural luster and take on a glassy appearance. Immediately upon thawing they become water-soaked. The leafy green tissues in the water-soaked area also have a dirty or muddy green color. In colorless or fleshy parts there is at first no discoloration. Later on the more sensitive tissues, especially the fibrovascular bundles, may become yellowish brown and finally black. In cabbage there is no such discoloration save possibly in the vascular ring of the stem. If the tissues have not been killed they soon lose the water-soaked appearance and look much as they did before, except that they may be more flaccid. If they are killed, discolorations usually develop which vary with the different kinds of vegetables and tissues. In tender, leafy tissues, such as cabbage, Italian broccoli, kale, and mustard, the epidermis often is loosened in spots, especially along the petioles, midrib, and ribs, so that it appears blistered and can be removed easily. Fleshy tissues such as turnips, radishes, rutabagas, and horseradish often show no discolorations, except in the vascular tissues, which become yellow, brown, or black.

In cabbage the outer leaves seem to be more resistant to killing by freezing than the inner leaves and the stem (pl. 6, *B*). Generally they thaw out without injury. After prolonged exposure to freezing temperatures, the inner tissues, especially the pith of the stem, usually are killed and soon become affected with bacterial soft rot.

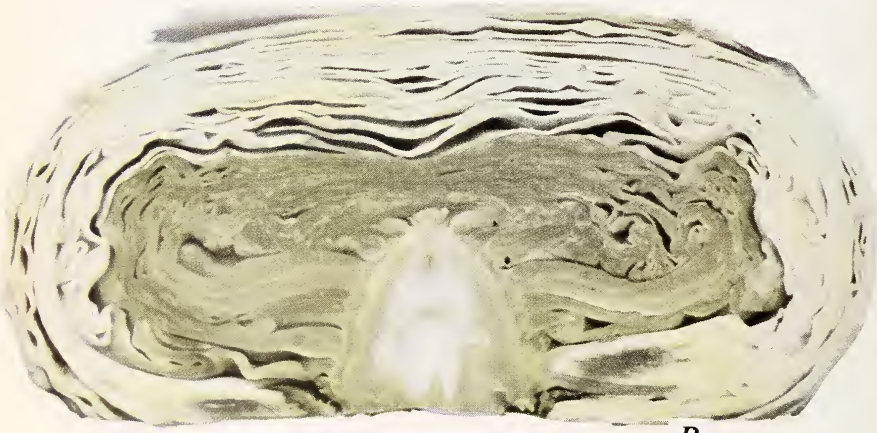
In dry air the killed tissues may dry out. Leafy tissues usually become brittle and parchmentlike while more fleshy ones become spongy and pithy or hard and stony. In moist air various rots develop, depending upon the external conditions. Thin, leafy tissues usually are affected by bacterial soft rot, alternaria rot, or gray mold rot. More fleshy ones are attacked by bacterial soft rots, blue mold rot, or rhizopus rot.

Control is obtained by avoiding exposure of the products to critical temperatures for a long enough period to cause their death (*63*, *71*, *74*, *87*).

Misc. Pub. 292, U. S. Dept. of Agriculture



A



B

A, Gray mold of cauliflower; *B*, freezing injury of cabbage.

GRAY MOLD ROT

(*Botrytis* spp.)

In cabbage, cauliflower, and turnip, as in most other vegetables, gray mold rot occurs most commonly in aging tissues. Consequently it is of most importance in these vegetables in storage where it is not infrequently the most important single factor of loss, since the causal organism can grow at cold-storage temperature. The rot is found even in active, freshly harvested stock if conditions are especially favorable for the fungus. Chief among these is high humidity. If this is associated with moderate temperature, the fungus attacks even the outer aging leaves of growing plants. Water-soaked, grayish-green areas are produced and usually a gray mold is present on the surface of affected tissues. In the more advanced stages of decay the grayish-brown mycelium and spore clusters of the fungus are characteristic (pl. 6, *A*). The most valuable diagnostic character is the fungus itself, which when sporulating cannot be confused with any other.

MOSAIC

The various types of mosaic disease that take such an enormous toll of many vegetable crops have not been serious on crucifers. White and black mustards, turnips, rutabagas, Chinese cabbage, brussels sprouts, cauliflower, and cabbage show varying degrees of susceptibility to mosaic. Fortunately, cauliflower and cabbage usually seem to show a natural resistance to this disease and in most regions are not seriously affected. A virus disease possibly related to mosaic is reported as becoming a serious factor in the growing of cauliflower in California. It has caused 20 to 30 percent loss in some fields, and apparently it is responsible for some of the excessive yellowing of the jacket leaves of cauliflower during transit.

The symptoms of mosaic in the crucifers are similar to those manifested in other crops. The characteristic mottled appearance of the leaves due to irregular patches of yellow or light-green and dark-green tissues, the crinkling and distortion of the leaf blades, and the stunting and yellowing of entire plants when severely infected are common manifestations of this disease.

In Oregon a mottling, yellowing, and dwarfing of horseradish plants appears to be due to a serious type of mosaic infection. The disease not only causes an underdevelopment of the roots, but also causes them to become rough, scaly, and pithy. Sometimes dark streaks occur in such diseased roots. These rather unusual manifestations of disease seriously affect the marketability of the roots (*11*, *16*, *42*, *64*, *68*).

PYTHIUM ROT

(*Pythium* sp.)

Pythium rot of cabbage has been found upon the markets occasionally, but so far it has been of no serious consequence. The causal organism is one of the soil fungi that frequently takes great toll of seedlings, and sometimes causes great loss in cucumbers, melons, string beans, and other products that come in contact with the soil

during rainy weather. Apparently conditions favoring the infection of head cabbage by this fungus are rare.

Affected cabbage heads usually show a water-soaked stump, and on cutting the heads it is found that the inner more or less compact head leaves are water-soaked for some distance out from the stalk (pl. 3, *B*). The affected tissues are moderately firm, but when pressure is exerted water may be expressed from the fleshy midribs. There is no unpleasant odor unless soft rot bacteria invade the affected tissues (*19*, *22*). (See Cucumber, Cottony Leak, p. 41.)

RHIZOCTONIA HEAD ROT

(*Rhizoctonia solani* Kühn (*Corticium vagum* Berk. and Curt.))

OCCURRENCE, SYMPTOMS, AND EFFECTS

Various crop plants belonging to the mustard family are subject to attacks by the common soil fungus *Rhizoctonia*. From a market point of view the head rot of cabbage and the root rot of turnips and radishes are the most important, but from the grower's standpoint the seedling diseases known as "damping-off" and "wire stem" are often the most important. Under favorable conditions one or more of the various types of disease induced by this fungus may occur in any of the vegetable-growing sections of the country.

When young plants are attacked they may be killed outright and are said to have "damped-off." Seedlings that have become well established before their roots and stems are seriously infected may continue to grow and produce spindling plants with tough discolored stems. This phase of the disease is known as "wire stem." Toward midseason the plants that escaped serious previous infection may show dark lesions on the stem and lower leaves, "bottom rot," or later dark-brown sunken lesions on the stem, midribs, and blades of the compact head leaves. This latter phase, known as head rot, is especially damaging to cabbages ready for market, because of the ability of the fungus to continue development during transit and while in storage. Heads so affected are also subject to bacterial infections that induce rapidly developing, foul-smelling decay.

The lesions induced by *Rhizoctonia* may usually be recognized by the moderately coarse cream-colored to brown surface mycelium and the irregularly shaped chocolate-brown sclerotia of the fungus. The affected tissues are light brown and semiwatery at first and change to dark brown or brownish black as the fungus extends deeper and the tissues dry.

The root rots of turnips, rutabagas, and radishes caused by *Rhizoctonia* are sometimes of serious consequence in the field, but in many instances most damage is done in storage stock. The fungus may enter the stem through leaf scars or injuries at the crown, or through infected rootlets. The lesions are usually circular in outline and light brown in color. As the fungus grows deeper into the tissues the affected area becomes dark brown and slightly sunken and the internal tissues change from a semiwatery to a spongy condition as water is lost by evaporation. Frequently there may be concentric rings of light and dark-brown tissues in storage turnips. Brown surface mycelium and sclerotia of the causal fungus may or may not be present.

CAUSAL FACTORS

The sterile form of the fungus (*Rhizoctonia solani*) is by far the most important cause of disease in plants. This stage of the fungus is ever present in the soil of most vegetable-growing districts, whereas the spore-forming stage (*Corticium vagum*) is rarely seen, and even when present on the surface soil and on the stems and lower leaves of plants it is of little consequence as a disease-producing factor. Under humid conditions the fine creamy-white mycelium and minute granular spores may be found on stems and the under sides of lower leaves of cabbages and other plants, but the spores do not survive long in dry air, and it is doubtful if they ever are important disseminating agents.

The rhizoctonia stage of this fungus is pathogenic to a great number of plants, and it is able to withstand adverse weather conditions remarkably well. It is able to survive in soils at freezing temperatures and below and is not killed at 100° F. The hard, compact masses of mycelium (sclerotia) may be expected to maintain the life of the fungus in practically any climate.

Several strains of this fungus are recognized, some being able to attack one plant, some another. The strain that causes bottom rot and head rot of cabbage will grow in culture at temperatures ranging between 48° and 91° F., but inoculation experiments indicate that the minimum for infection of cabbage is near 53°, the optimum 77° to 80°, and the maximum 88° to 90°.

For growth in culture the strain of *Rhizoctonia* which causes rot of turnips has its minimum at about 35° F., optimum 73°, and maximum 89° to 94°. Inoculations with this fungus indicate that the minimum temperature for infection of turnips is about 35°, the optimum 66° to 77°, and the maximum between 84° and 90°.

Except for very low or extremely high temperatures the most important limiting factors in determining infection and decay by *Rhizoctonia* are soil moisture and humidity. Since this fungus does not require wounds for producing infection it may attack practically any part of a plant with which it comes in contact, providing there is enough moisture to permit it to grow on the surface tissues long enough to gain entrance into them. Once the hyphae have invaded the tissues they will continue to develop and cause decay regardless of external moisture, although high humidity always favors rapid development of decay. In storage stock high humidity favors infection and decay; low humidity limits both.

CONTROL MEASURES

Control measures for reducing the amount of infection by *Rhizoctonia* have not been very successful because the fungus is already present in most soils. By sterilizing the soil of the seedbeds and spraying the young seedlings with bordeaux mixture the early types of infection may be greatly reduced. All seedlings showing discoloration of the root system or stems should be discarded at transplanting time.

Cabbages, turnips, rutabagas, or radishes that show severe rhizoctonia infection should not be shipped or stored. If necessary, however, slightly affected products may be stored for a month or so

without excessive loss, if the temperature is held at 32° to 34° F. and the humidity is kept as low as possible without causing wilting (*43*, *79*, *84*).

RHIZOPUS SOFT ROT

(*Rhizopus nigricans* Ehr.)

The members of the mustard family are often subject to infection and decay by *Rhizopus*. This fungus is ever present in the air and soil, and consequently there is always more or less danger from infection if conditions are favorable. Excessively wounded or bruised stock is especially susceptible to infection if warm, humid conditions prevail. This disease is of little or no consequence in the field or on leafy crops such as mustard and kale, but with the more fleshy vegetables such as cabbage, cauliflower, turnips, and rutabagas sometimes considerable loss occurs on account of decay developing during transit and marketing. The chief predisposing factors are wounds and high temperature.

The decay in succulent tissues is light brown, soft, and watery; in root crops the rot is moderately soft and moist, but it lacks the disagreeable but characteristic odor of the bacterial soft rots that sometimes present a similar appearance. Usually in cabbages, cauliflower, brussels sprouts, and turnips the coarse, stringy mycelium bearing white and black sporangia make the diagnosis of this disease fairly easy.

Rhizopus does not affect cabbage, cauliflower, or root crops in cold storage (32° F.) The minimum temperature for the development of rhizopus rot is about 44° F., the optimum between 77° and 82°, and the maximum about 90° (*79*).

SUNSCALD

In cabbage, scald usually occurs only on leaves that are exposed on the top of the head. The first symptom is a water-soaked or even blistered appearance of an irregularly shaped area, which is promptly followed by drying out, so that a bleached, papery, or parchment-like island surrounded by healthy tissue is the result.

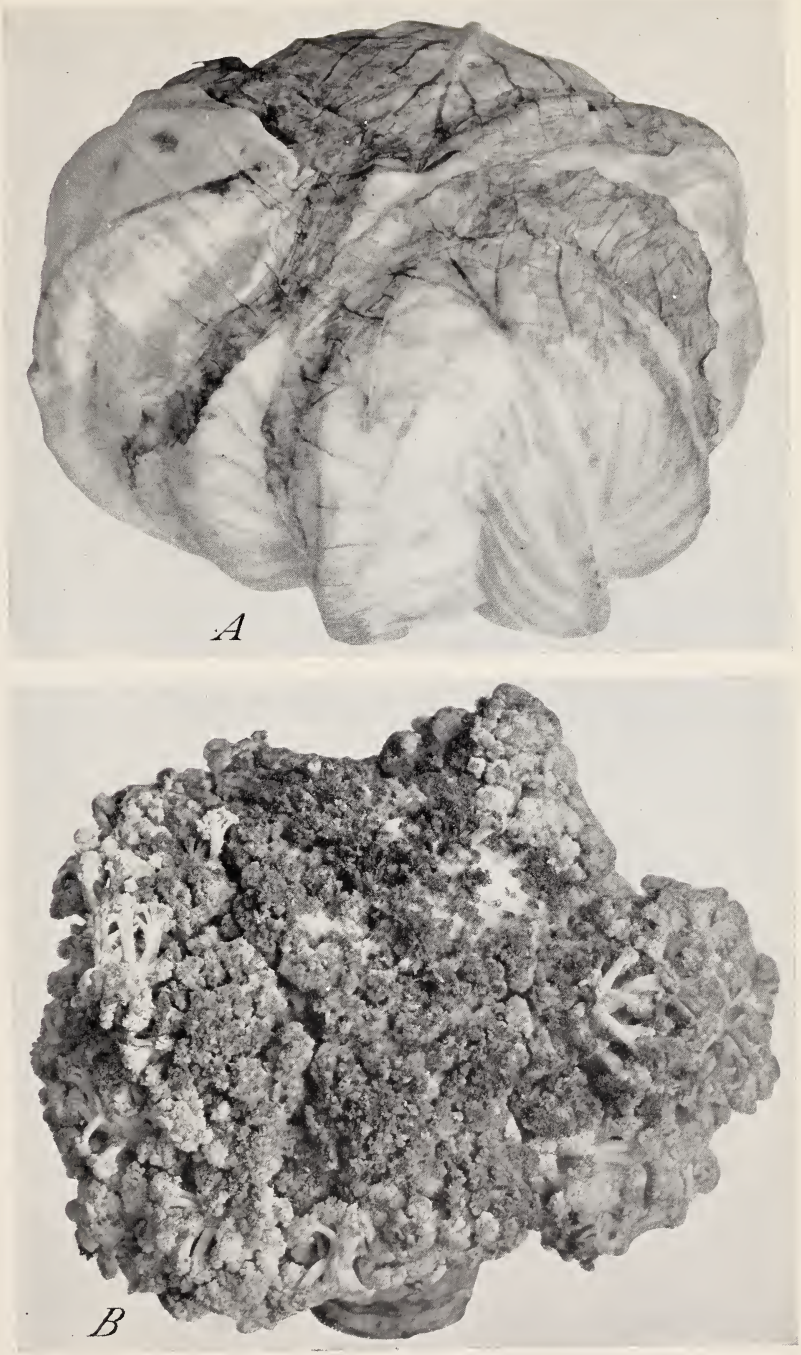
The scalded area either remains parched and bleached or is invaded by wound parasites and saprophytes, chief among which are the bacteria that cause soft rot and such fungi as *Rhizopus* sp. and *Alternaria* sp.

In the field not much can be done to prevent this injury; in gardens and in greenhouses it is advisable to time sprinkling so that the hot sun will not shine on the wet tissues.

TIPBURN

Tipburn of cabbage is most common in the Northern States. It frequently causes much damage to cabbage grown in soil deficient in potash and comparatively high in nitrogen. In seasons of severe drought, death, and discoloration of the leaf margins sometimes occur which in effect is also a type of tipburn.

Characteristic symptoms of this disease are yellowing, browning, and gradual drying of the leaf margins. Frequently the outer leaves are the only ones affected and these may be trimmed off before the



A, Tip burn of cabbage; *B*, brown rot of cauliflower.

cabbages are marketed, but in some instances the tips of the leaves a fourth of the way through the head may show yellowish or grayish-brown discoloration (pl. 7, *A*). Cabbage showing tipburn is greatly discounted on the market because of appearance and the loss entailed in preparing it for table use. Affected stock whether in the field, in transit, or in storage is also subject to invasion by decay-producing organisms, such as bacteria, *Alternaria*, *Rhizopus*, and *Botrytis*.

Control of tipburn has been obtained by application of potash fertilizers to some soils; to others a heavy dressing of stable manure has been found beneficial (*8*, *71*).

WATERY SOFT ROT (SCLEROTINIA ROT)

(*Sclerotinia sclerotiorum* (Lib.) DBy., and other *Sclerotinia* spp.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Among the most serious diseases of vegetables are those caused by soil organisms. Watery soft rot is caused by a soil fungus which is more or less common in all trucking regions, and is especially destructive because it can attack such a wide variety of crops. Practically all vegetables except onions and potatoes are subject to this disease. A characteristic soft, watery decay is produced and affected tissues are often covered with a white cottony mold (pl. 2, *B*). In transit, storage, and on the market badly decayed products actually leak, but there is no disagreeable odor like that produced by many other diseases. The degree of wetness varies considerably with the succulency of the tissues and the humidity of the surrounding air. In dry air the water frequently evaporates as fast as it is liberated and the tissues dry down into a mummy or into parchmentlike remnants. Succulent tissues in moist air generally are completely liquefied, so that a pool of juice with the characteristic odor of the plant tissues is all that remains. The color of the lesions varies widely with various hosts and individuals. When the lesions are moist they appear water-soaked, with little change in color. Frequently the borders of the lesions in white or cream-colored tissues are pink (hence the name "pink rot" of celery, cabbage, and cauliflower), whereas the inner portions are brownish.

In the field the disease generally occurs in the basal part of plants or in the plant parts near or lying on the soil. It frequently destroys the cortical part of the stem or the basal part of the lower leaves of plants, causing the wilting or dropping of the leaves and total collapse of plants. Lettuce and cabbage drop and celery foot rot are typical examples. Frequently the disease manifests itself as damping-off. However, the fungus is by no means limited to the basal parts of plants; it can attack the floral parts 3 to 4 feet above the ground.

In storage and in transit the fungus frequently makes so vigorous and compact a growth as it spreads from one individual to another, enmeshing them in its mycelium, that so-called nests result. These are particularly frequent in hampers of beans and peas.

The fungus furnishes a good means for identifying the disease in the field, in transit, and in storage under moist conditions. It usually develops a luxuriant white, cottony mass of mycelium, which is so characteristic that the disease sometimes is known as cottony rot.

Even more characteristic are the large resting bodies or sclerotia, which form when the fungus begins to dry out or when the food supply begins to wane. These are white at first and later turn bluish to black. In storage stock large sclerotia are often formed on affected cabbage, celery, carrots, and turnips. Frequently in the field they literally fill the pith region of the stems of the sunflower, lettuce, celery, and cabbage.

CAUSAL FACTORS

The organism that most commonly causes the disease is the fungus *Sclerotinia sclerotiorum*. Other species of *Sclerotinia*, especially *S. minor* I. C. Jagger and *S. intermedia* G. B. Ramsey, cause similar rots, which cannot be distinguished from that caused by *S. sclerotiorum* without a study of the causal organism.

These fungi persist in the sclerotial stage in the soil. As soon as moisture and temperature conditions are favorable, the sclerotia may develop an abundant mycelium, which readily spreads to young plants or any other source of food. If moisture, temperature, and light are favorable, the sclerotia buried in the soil may send out diminutive mushroomlike growths, the apothecia, in the cups of which are borne great numbers of spores, which, upon attaining maturity, are forcibly shot out into the air. If these spores alight on moist surfaces, they germinate, and, if food is available, they either cause direct infection of plants or develop new colonies of the fungus from which it can spread and cause infection by means of the mycelium.

The chances, therefore, are very great that watery soft rot may develop in transit or storage in stock which appeared sound but which came from a field infested with the fungus. Plants taken from fields free from *Sclerotinia* will not develop the rot unless they come in contact with other diseased plants or soil contaminated with the fungus. A few infected specimens in a package, however, may be sufficient to cause complete decay of the entire contents. Although the fungus does not need wounds for entry, wounded tissues become infected more readily than those free of wounds.

Appearance of the disease in the field, in transit, and in storage depends upon moisture and temperature conditions. Moisture is the most important limiting factor for the germination of sclerotia, the spread of the fungus, the germination of the spores, and infection by mycelium and spores. Dry conditions, which prevent formation of apothecia, and dry plant surfaces when the spores are discharged are the most important factors in limiting the onset of the disease.

High temperatures (above 80° F.) prevent formation of apothecia or at least spore formation. Without spore discharge the distribution of the disease in a field is limited. The spores can germinate at temperatures ranging from 39° to 88°. After the fungus has once become established in a field following spore discharge, temperature is a secondary limiting factor for infection, because the fungus has a wide temperature range. It can grow and cause infection at 30°.

String beans inoculated with *Sclerotinia libertiana* Fckl. and held at 32° for 26 days showed penetration of one-fourth inch by the fungus. Inoculations made with *S. minor* and *S. intermedia* under the same conditions caused decay which extended 1 inch and 1½

inches, respectively, lengthwise of the pods. Growth of the fungus, amount of infection, and severity of the rot increase rapidly with rising temperatures until the optimum temperature for growth and infection is reached between 70° and 78°. Above this temperature they drop rapidly. At 88° to 90° there is practically no growth or infection. Under the most favorable temperature and moisture conditions, the first symptoms may appear within 6 to 48 hours after inoculation, and the tissue may be completely destroyed in 3 to 5 days. The fungus can withstand a temperature as low as 23° and remain viable.

After infection has taken place the rate of development of the lesions is less dependent upon moisture than upon temperature. For this reason severe losses are commonly noted in transit and storage at temperatures between 32° and 45° F. If the stock comes from an infested field and is wet when put into transit or storage, or if it is put into contact with stock already affected, low temperatures will slow up the development and spread of the rot but will not control it completely.

CONTROL MEASURES

No measures are known which will completely control the disease in the field when temperature and moisture conditions are favorable for the fungus.

It is very desirable to destroy diseased plants and plant trash which give the fungus a chance to propagate and in which the resting bodies (sclerotia) are formed. A well-aerated soil is likely to have less of the fungus than one that is not well aerated.

Affected products should not be packed or stored with sound.

Although low temperatures do not completely control the disease, they retard its development so much that precooling and transit refrigeration are highly desirable (*17*, *57*, *60*, *62*, *79*).

YELLOWS

(*Fusarium conglutinans* Wr.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Yellows is a very serious field disease of cabbage and occasionally of cauliflower, kale, kohlrabi, and turnip. It is a vascular disease of the roots and stem which causes secondary symptoms in the top and frequently the death of the plant. Although it frequently causes field losses as high as 50 to 90 percent in susceptible varieties during favorable weather conditions, it is only indirectly a market factor. In kohlrabi and turnip the lesions in the marketable tissues may cause it to be of direct market significance. Affected cabbage plants, when they head at all, are ill-shaped and poorly colored.

The disease occurs in those cabbage-growing sections of the United States in which hot, dry weather occurs during the cabbage-growing season, especially after transplanting (*76*). For this reason it is a serious factor only in the central cabbage belt extending from Kansas to the Atlantic coast with its northern limits in southern Minnesota, central Wisconsin, and southern New York. Yellows is of no importance in the Southern States because cabbage is grown there as a winter crop. It does not occur in Europe.

CAUSAL FACTORS

The causal fungus, *Fusarium conglutinans*, lives in the soil, from which it invades the fibrous roots of the plant. There is evidence that once it has become well established, it may persist indefinitely in a favorable soil. Continued cropping of a soil with members of the mustard family increases the abundance of the fungus so enormously that cabbage culture ceases to be profitable. It may be introduced into a disease-free soil by diseased plants from the seedbed, by water, by wind blowing the dust from field to field, by animals, and by farm tools.

Whether or not the disease will be a serious factor when the soil is infested is determined primarily by temperature. Although yellows may develop at temperatures as low as 53° to 55° F., no appreciable development takes place if the soil temperature is below 61° to 63°. The severity of the disease increases with increase of temperature above this point up to 82°, this temperature being most favorable for its development.

Once infection has occurred, dry weather and dry soil are more favorable for the disease than a wet season or a wet soil.

CONTROL MEASURES

The only practicable method of control is the use of resistant varieties. The first yellows-resistant strain was Wisconsin Hollander, selected from the winter variety, Hollander. A little later, selections made from two standard sauerkraut varieties led to the two yellows resistant varieties Wisconsin All Seasons and Wisconsin Brunswick. Other important resistant strains now available are the midseason varieties, Marion Market, Globe, All Head Select and Iacope, and the early variety Jersey Queen. There is promise that resistant strains can be secured by selection from any of the standard market types (*3*, *23*, *25*, *38*, *40*, *67*, *72*, *75*, *78*).

CHINESE CABBAGE

The Chinese cabbage, or petsai of the market (*Brassica pekinensis*), is a member of the mustard family and is related to the common cabbage. It does not form compact heads, but a more or less open, soft mass of leaves. The factors governing the marketability of Chinese cabbage are essentially the same as those of the common cabbage (p. 3). The most important market diseases are bacterial soft rot, black rot, and black leaf speck (p. 8) (*5*, *17*, *42*, *64*).

BRUSSELS SPROUTS

The brussels sprouts of the market are the buds of a member of the mustard family (*Brassica oleracea* var. *gemmifera*). Desirability of the buds depends primarily upon crispness, tenderness, and freedom from blemishes and decay. Essentially the same diseases that affect cabbage and other cruciferae may be found on the plant in the field and the buds after harvest. The most important market and transit diseases are bacterial soft rot (p. 5) and alternaria leaf spot (p. 4).

CAULIFLOWER

The cauliflower of the market is the malformed condensed flower head with short, fleshy branches of a plant (*Brassica oleracea* L. var. *botrytis*) very closely related to the cabbage. By breeding and selection, strains have been developed which produce firm, smooth, white, well-rounded flower heads or "curds," and these are greatly in demand on the market. The cauliflower is very exacting as to climatic conditions; consequently, it is grown successfully on a large scale in only a few localities. In order to produce the white heads demanded on the market, it is necessary to protect the young heads from the sun by folding and tying the large leaves over them, or providing some other covering so that development of the chlorophyll is stopped. Great care must be exercised to cut the heads at the right stage of maturity, otherwise the floral parts develop. Waiting too long is disastrous also because later the heads break up and become rough due to the elongation of the flower stalks.

In the best cauliflower the outer leaves ("jackets") are green, the enclosed head is creamy white to white, and the bud clusters are crowded closely together forming an even, firm head. Good heads will remain compact for a short time in the field, and under proper conditions will carry well in transit, maintaining good marketable condition for some time. Some strains of cauliflower have a tendency to flower very rapidly. The branches of the head elongate, become slender and green, thus producing an unsalable head. This type of head should never be cut, since once the elongation has started it will continue under transit and market conditions.

Development of the colorless leafy bracts in the flower head leads to a condition known as "fuzzy," "barber," and "old man." When the heads begin to spread so that the individual buds are separated, the condition is referred to as "riceyness." Unless these conditions are severe they do not very materially lower the selling price of the cauliflower. Heads cut just on the verge of fuzziness or riceyness, even though these conditions are not apparent at the time of cutting, will develop them in transit unless immediately refrigerated. Material for such growth of the cut head is furnished by the heavy leaves around the head. Another type of spreading or opening is caused by excessive shrinkage due to water loss. This occurs in stock that has been held for a long time, while the spreading due to overmaturity takes place within a few days after the stock is cut.

Since the market demands green jackets, any disease that spots the leaves or that causes premature yellowing or dropping is important from the market point of view.

While the term "broccoli" is sometimes applied to winter cauliflower, as grown in the Pacific Northwest, it is now more generally used in referring to the green, loosely formed floral heads and associated tender leaves and stems of the plant *Brassica oleracea* (var. *italica* Plenck). This flowering or sprouting broccoli has become very popular in recent years. Although this plant is subject to many of the diseases found on cauliflower, so far few diseases of market importance have been encountered. Bacterial soft rot (p. 5) and injury due to aphids are the most serious market troubles.

Cauliflower is subject to the same diseases that affect cabbage and other members of the Cruciferae (p. 8). Some of these diseases are

especially important in cauliflower. Those that either blemish or decay the curd are more important than those affecting the leaves primarily. The important diseases are: Bacterial soft rot (p. 5), black rot, bacterial leaf spot, brown rot, clubroot, gray mold rot (p. 17), mosaic (p. 17), ring spot, and watery soft rot (p. 21) (*10*, *68*, *74*, *80*, *81*).

BACTERIAL LEAF SPOT

(*Bacterium maculicola* (McC.) McCulloch)

Bacterial leaf spot is a minor disease of cauliflower and cabbage found occasionally in the field and on the market. It is most important on cauliflower grown in the San Francisco Bay district in California, but it has also been reported from Virginia, Florida, and New York.

The disease disfigures the leaves of cabbage and cauliflower as well as the curds of the latter, thus reducing the market value. It also opens the way for invasion by the soft-rot-producing organisms.

The disease is characterized by abundant specking, spotting, and blotching of the green leaves, both on and between the veins, as well as on the flowers. On the latter the lesions appear as small gray to brown spots on the epidermis and deeper tissues of the buds and branches of the curd. The affected tissues are firm at first but later become soft as secondary organisms invade the lesions. The spots on the leaves at first appear water-soaked and then brownish to purplish gray. In transmitted light they appear thin and almost colorless in the center with dark borders. The lesions usually are very small and are circular when young, but soon become more or less angular as they reach the veins. The spots often coalesce, thus becoming elongated and irregular, giving the leaf a ragged appearance. Severely affected leaves turn yellow and fall prematurely.

The causal organism, *Bacterium maculicola*, apparently enters only through open stomata; consequently, it does not attack very young or old leaves, nor does it readily attack the bleached parts of plants. The spots are most abundant on the lower side of leaves. In California it has been observed that the disease is more likely to occur on cauliflower curds that are slightly overmature and on those showing slight injuries. The tender floral parts injured by cold weather are also susceptible to invasion by the bacterial spot organism. The period of incubation is 3 to 6 days. The pathogen is favored by moderately cool weather, making its best growth at 77° F. It does not grow at temperatures above 86°, and it is very sensitive to dry air and sunlight. High humidities are necessary for infection.

Nothing is known relative to control measures. The causal bacterium has been found in the soil in California and on the body of the red-bordered stinkbug, thus indicating that this insect may act as a disseminating agent (*26*, *47*, *66*, *pp. 300–313*).

BITTER BROWN ROT (BROWNING)

A break-down and discoloration of the pith of stems and heads of cauliflower (called bitter brown rot or browning)[3] grown in certain districts of New York State has caused considerable loss to cauliflower crops since 1918.

[3] CHUPP, C., and HORSFALL, J. G. BROWN ROT OF CAULIFLOWER. U. S. Bur. Plant Indus., Plant Disease Reptr. 17: 157–159. 1933. [Mimeographed.]

Fluctuation in soil moisture, as well as a deficiency of boron in the soil, appear to be important factors in causing this trouble. It has been noted that if the season is relatively dry at heading time no trouble is experienced, but if the first part of the season is dry and the latter part wet, serious damage due to browning is likely to occur. The disease is generally less prevalent on well-drained soils. An acid soil condition seems to favor the development of bitter brown rot, consequently it is sometimes beneficial to add borax or a heavy application of manure to some soils for control of this trouble.

The first noticeable symptom of bitter brown rot is often a slight brownish discoloration of the flower head or curd. On examination it is usually found that the stem or branch leading to the discolored part is also brown and the pith may be in a state of dissolution. Although the entire head may become brown, it remains firm until bacteria or other secondary decay-producing organisms enter. The tissues of affected heads are very bitter to the taste, but there is no disagreeable odor until bacterial soft rot develops.

This physiological break-down of cauliflower is similar in some respects to "mottled heart" or "brown heart" of turnips and rutabagas (Turnips, p. 34 (*17a*, *32*)). In New York the addition to the soil of 5 to 25 pounds of borax per acre has given good control of this trouble.

BROWN ROT

(*Alternaria brassicae* (Berk.) Sacc.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Brown rot is caused by the same organism which causes black leaf spot or black mold rot on other members of the cabbage family. The lesions on curds of cauliflower, however, are known as brown rot. Although the disease may kill seedlings it is not generally severe in the field because usually old or dying leaves alone are affected. The importance of the disease lies in its effects upon the curd in transit and in storage.

On the curd the fungus causes a water-soaking and browning of individual buds or groups of buds (pl. 7, *B*). The color of the lesions varies with the age. At first they are light brown, darkening with age to nearly chestnut brown, followed by an olivaceous color when the dark-colored spores of the fungus develop.

Sometimes a curd is spotted with innumerable minute spots and at other times with large blotches. The lesions may be quite dry and firm or they may be wet and soft so that they are readily confused with the brown spots caused by bacteria. Often the brown-rot lesions are invaded by the soft-rot organism.

On leaves and stems the symptoms are essentially those found on other members of the cabbage family. They appear first as small dark-brown to black spots about one-twentieth of an inch in diameter. With temperature and humidity conditions unfavorable to the fungus they may become no larger. The ultimate size and shape of the lesions depend also upon the nature of the tissue attacked. On the leaf blade the lesions may remain small (about one-twentieth of an inch) and dark brown, or they may enlarge into circular lesions one-fourth to one-half inch or more in diameter and grayish to brown in color. The fruiting fungus may appear in the form of scattered

minute black areas on these spots. Sometimes the spots coalesce to form large irregular lesions, which may involve more than half of the leaf. Between the green of the healthy leaf tissue and the brown of the lesion there is a zone of yellow. On the veins, petioles, and stalks the lesions are elongated parallel with the veins but are otherwise like those of the leaf lamina.

When the humidity is high the fungus often penetrates completely through the tissues. As a result the stems of seedlings are frequently broken off by cankers. When moisture conditions are favorable the lesions on the leaves become covered with the sporulating mycelium of the fungus so that they appear olivaceous. If the lesions are numerous on the older leaves, these leaves will turn yellow prematurely and fall off.

CAUSAL FACTORS

The causal fungus, *Alternaria brassicae*, lives over winter in the soil and in the debris of infested plants of the previous season. The spores of the fungus also live over on the seed. Seeds often become infected in the pod, because the fungus attacks and penetrates the pods. From these sources the seedlings become infected, and on them are produced the spores that serve as inoculum for other plants. The seedlings can serve as a means of carrying the disease from the seedbed into the field.

The fungus produces infection of the heads through a wide temperature range. At about 35° F. spots appear after 5 to 7 days and are about one-twentieth of an inch in diameter after 14 days. The rate of development of the lesions increases with increasing temperature up to about 86°. At temperatures between 78° and 86° lesions one-twentieth of an inch in diameter develop within 24 hours after inoculation and the rot penetrates about one-fourth inch in 7 days. At temperatures between 32° and 34°, with high humidities (74 to 100 percent) lesions are formed, but they do not penetrate deeply. These data show that it is possible to harvest cauliflower, which appears free from brown rot but which may develop visible lesions during transit, depending upon the temperature.

CONTROL MEASURES

Although the temperatures obtained in good refrigerator cars (40° F.) will not entirely stop infection or development of the disease they do give practical control for about 2 weeks. It is desirable to keep the curds as dry as possible (*80*, *82*). Since temperatures in transit cannot be maintained low enough to absolutely control the disease, field control becomes of special importance. Seed treatment with hot water, 132° for 10 minutes, has been found effective. Spraying with bordeaux mixture 4–4–50 with the addition of a sticker is useful in severe outbreaks.

RING SPOT

(*Mycosphaerella brassicicola* (Fr.) Lindau)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Although ring spot may affect various plants belonging to the mustard family it seldom is of economic importance except on cauli-

A, Ring spot of turnip leaves; *B*, greatly enlarged ring spot lesions of cauliflower. Note the great number of black fruiting bodies over the spots.

flower grown in California and Oregon. Cabbage, brussels sprouts, kale, and rutabagas are sometimes spotted by this disease, but radishes and white turnips have not been found affected.

Most damage produced in the field is due to premature yellowing of the leaves and subsequent defoliation, which in some instances results in reduction in size of the heads. However, under good growing conditions even badly affected plants may produce good heads. From the market point of view the disease is important because badly spotted and yellowed leaves greatly injure the marketability of affected stock even though the heads are satisfactory.

Practically all parts of a plant are susceptible to infection. The lower leaves are most often severely attacked, the younger ones being least susceptible. The first visible infections appear as small circular spots having a dark center surrounded by a greenish-yellow water-soaked border. These spots are about one-thirty-second of an inch in diameter and are visible on both sides of the leaf. As the spots enlarge they usually maintain the circular outline and have light-brown to grayish-brown centers surrounded by a narrow olive-green or olive-gray border (pl. 8, *A*). When the leaves turn yellow, the spots retain their greenish borders and become very conspicuous against the yellow background. The black pycnidia and perithecia may appear in concentric rings toward the outer part of the central region or be scattered promiscuously over it. The pycnidia and perithecia are sometimes so abundant that the whole spot appears black (pl. 8, *B*).

CAUSAL FACTORS

The causal organism (*Mycosphaerella brassicicola*) lives over from season to season on the old leaves, stems, and seed pods of infected plants and apparently lives for some time in the soil. Moderately low temperatures and high humidity are necessary for the spores of this fungus to germinate and produce infection. Spore germination is slow at 36° F. but rapid at temperatures ranging between 60° and 72°. Experiments have shown that the ascospores may germinate and produce infection within 18 to 30 hours at a temperature of 68° to 72°. After infection has taken place it is 2 to 3 weeks before the spots have developed sufficiently to make a definite diagnosis of ring spot possible. The lesions are usually about a month old before the small black dotlike fruiting bodies become evident.

Although spots on the leaves of cauliflower in storage at 32° F. may increase in size slightly within 2 weeks, there is no marked increase unless the temperature reaches 42° to 48° or higher. Under refrigeration it is unlikely that any appreciable development of old spots will take place, and experimental evidence tends to show that few, if any, new spots will form. At room temperature (65° to 75°) ring spot lesions have been observed to increase in diameter as much as one-eighth of an inch within a week.

Much of the yellowing and dropping of leaves of cauliflower during transit and storage, which has been heretofore attributed to the effects of ring spot, is apparently due to temperature. Many of the leaves of freshly harvested cauliflower placed in storage at 45° F. have been observed to turn yellow and drop within a week regardless of whether they are infected. At 41° the rate of yellowing of the jacket leaves

is much slower, and at 32° they do not change much in color within 30 days.

CONTROL MEASURES

The following control measures are recommended: Immerse the seed in hot water (122° F.) for 30 minutes and follow seedbed-sanitation measures. Prompt and effective refrigeration is important for the harvested crop.

Spraying and dusting have not been found effective, and they injure the plants. No resistant strains of cauliflower or broccoli are known and all varieties tested so far appear about equally susceptible (*81*).

COLLARDS AND KALE

Collards and kale are horticultural varieties of a nonheading type of cabbage plant (*Brassica oleraceae* var. *viridis*). Both are subject to many diseases that occur on cabbage and other members of the mustard family. In transit and on the market, bacterial soft rot (*Bacillus* spp., p. 5) is the most important disease. Yellows (*Fusarium conglutinans* Wr., p. 23) is a market factor at times because of the discoloration of foliage associated with it (*40*).

CRESS

The name "cress" is used for several species of crucifers that are used in salads and for garnishing. The leaves of the common garden cress (*Lepidium sativum*) and water cress (*Radicula nasturtium-aquaticum*) are most often found upon the market. The marketability of cress depends upon the color, tenderness, crispness, and freedom from blemishes and decay of the foliage. Few diseases affect this group of plants while they are young enough to be marketed as cress. Bacterial soft rot (p. 5) causes some damage in badly wounded and bruised stock during transit and marketing. Damping-off (*Pythium debaryanum* Hesse and *Corticium vagum* Berk. and Curt.) and a stem rot or sclerotinia rot (p. 21) sometimes affect plants in the greenhouse (*29*).

HORSERADISH

The horseradish of the market is the fleshy white taproot of the horseradish plant (*Radicula armoracia*), a member of the mustard family. The marketability of this product depends chiefly upon the size and smoothness of the roots and freedom from growth cracks and bacterial root rot. The horseradish plant is remarkably free from serious diseases. Bacterial root rot (*Bacterium* sp.) is the only disease of market importance. The alternaria leaf spots (p. 4), the bacterial leaf spot (p. 26), mosaic (p. 17), and white rust (p. 33) reduce the vitality of the plant somewhat in severe cases of infection and indirectly influence the marketability of the roots. Rhizoctonia rot (p. 18) and black root rot (*Thielaviopsis basicola* (Berk.) Ferraris) affect not only the fibrous roots, thus interfering with the nutrition and in severe cases causing underdevelopment of the taproot, but also the taproot itself, and consequently are of both indirect and direct market significance (*16*, *48*).

BACTERIAL ROOT ROT

(*Bacterium* sp.)

In bacterial root rot, which has been reported from New Jersey, the central cylinder of the root is invaded by the bacteria. The affected tissue is water-soaked at first, later turns yellowish in color, and may finally become soft and disappear, leaving a hollow in the center of the larger roots. The disease develops and spreads in storage if the air is humid and the temperature is high. Affected roots are subject to secondary infection by decay-producing organisms. They are of little market value and are undesirable for planting stock. It is suggested that roots intended for planting be trimmed carefully and treated with mercuric chloride (1 ounce to 8 gallons of water) for 15 minutes, then rinsed and dried before planting (*58*, *59*).

BLACK ROOT ROT

(*Thielaviopsis basicola* (Berk.) Ferraris)

Black root rot occurs on a wide range of hosts. In most of them it is of indirect significance from the market point of view, because the fungus causes lesions only on the underground parts. In the horseradish, however, the disease is a factor because the marketable product is directly affected.

The fungus attacks the cortical tissues of the roots and the underground portions of stems and causes roughening and a charcoal-black discoloration, which sometimes extends above the ground. Within, the tissues are tinted red or pink. The lateral or fibrous roots frequently are badly rotted in some plants so that only a stub of the root system remains. The plants may merely be stunted or the root system may be so severely affected that the plants die while young. Affected plants frequently recover. The above-ground parts are often dwarfed and yellowed.

Thielaviopsis basicola is one of the soil fungi. It overwinters in the soil in debris of affected plants. It can grow through a wide temperature range, 50° to 104° F., but does not cause root rot when the soil temperature is above 75°. Consequently, it is of importance only during cool seasons or in sections having a cool climate. The fungus is favored by an abundance of moisture.

For control of this disease, sterilization of the seedbed and rotation of crops have been recommended (*34*, *35*).

KOHLRABI

The kohlrabi of the market is the swollen, fleshy stem of *Brassica oleracea gongylodes*, a member of the mustard family. The stem usually is marketed with the leaves attached. The most important factors determining its marketability are tenderness, absence of woodiness, and freedom from blemishes and decay. The leaves also must have green color and must be free from blemishes and decay.

The plant is subject to many of the diseases that attack cabbage, but few of these are direct market factors. Bacterial soft rot (p. 5) is the most important disease. Discoloration in the stem due to black rot (p. 10) occurs occasionally and affects the marketability of the stem.

MUSTARD

The mustard plant (*Brassica* spp.) is grown commercially for two purposes—for the seeds, which yield oil and are used as a condiment, and for the leaves, which are used for "greens." *Brassica nigra* and *B. alba* are the best seed-producing varieties; *B. juncea crispifolia* Bailey is usually planted for greens. The marketability of mustard for greens depends upon good green color of the leaves and freedom from blemishes and decay.

The mustards are not often seriously affected by disease, although they are susceptible to most of the diseases which attack other crucifers (p. 3). The marketability of mustard is most generally affected by such diseases as alternaria leaf spot (p. 4), bacterial soft rot (p. 5), cercospora leaf spot (*Cercospora* sp.), downy mildew (p. 13), mosaic (p. 17), powdery mildew (*Erysiphe polygoni* DC.), and white rust (p. 33), which affect the above-ground portions of the plants. The marketability of the leaves and the production of seed pods are sometimes seriously affected by clubroot (p. 12), which causes malformation and decay of the root system.

RADISH

The radish of the market is the fleshy taproot and hypocotyl of the radish plant (*Raphanus sativus*), a member of the mustard family. Although only the root is eaten, radishes are marketed in bunches with the tops attached. Consequently the appearance and condition of the tops as well as of the roots are market factors. The main market requisites for the root are tenderness, crispness, and freedom from pungent taste, blemishes, and decay. The tops must be green and likewise free from blemishes and decay.

The leaves are affected with bacterial soft rot (p. 5), bacterial spot (*Bacterium vesicatorium* var. *raphani* White), downy mildew (p. 13), powdery mildew (*Erysiphe polygoni* DC.), and white rust (p. 33). These affect not only the leaves, but, indirectly, the quality of the roots. Bacterial soft rot is by far the most important.

The roots are affected with ascochyta root rot (*Ascochyta* sp.), bacterial soft rot, black root (*Aphanomyces raphani* Kend.), clubroot (p. 12), and rhizoctonia rot (p. 18).

BLACK ROOT

(*Aphanomyces raphani* Kend.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Although black root has been reported most often on midwestern markets and in the home gardens planted to the White Icicle variety of radish, it has been also noted in numerous sections of the country, but it is always most serious on the long varieties. Globe radishes are generally resistant to infection. The disease is usually more severe on late plantings than on early ones.

Black root is primarily a disease of the roots, but seedlings are sometimes affected. The first symptoms of this disease are small grayish black surface discolorations about the base of the secondary roots. As the fungus penetrates into the main root, dark patches become visible under the epidermis. Later the epidermis itself is blackened and

A, Black root of radish; *B*, downy mildew discoloration of turnip root.

eventually numerous discolored spots may coalesce, forming black areas covering a large part of the root or entirely girdling it (pl. 9, *A*). Early infections often lead to various types of malformations as the roots develop. Extensive surface discoloration and longitudinal cracking of the roots entirely destroy radishes for marketing. The affected tissues are firm and do not disintegrate unless there is secondary infection by bacteria of the soft-rot group.

CAUSAL FACTORS

The causal organism, *Aphanomyces raphani* Kend., is a soil-inhabiting fungus that is able to infect radish roots through the natural wound made where the secondary roots emerge through the tissues of the primary root. Infection may take place through other wounds if available, but experimental evidence indicates that about 95 percent of the natural infections are made through the ruptured tissue at the base of the emerging secondary roots.

In culture this fungus grows throughout a wide range of temperatures, although low and very high temperatures inhibit its development. No growth takes place at 48° or at 95° F.; most rapid growth is made between 73° and 81°. The oospores of this fungus develop most abundantly between 68° and 77°.

CONTROL MEASURES

Since the White Icicle and other long white varieties are most susceptible to black root, sometimes in heavily infested soils it is advisible to change to globe types of radishes which are rather resistant to this trouble. Early planting to avoid high soil temperatures, which favor the growth of the fungus, is sometimes helpful.

The organism lives over from year to year in the soil and may be scattered in infested soil. Fortunately, no other crop plants seem to be affected, so that rotation of crops together with soil sterilization may be of value.

Of the numerous varieties tested for resistance, only the White Chinese has given satisfactory results (*20*, *39*).

WHITE RUST

(*Albugo candida* (Pers.) O. Kuntze)

OCCURRENCE, SYMPTOMS, AND EFFECTS

White rust is found all over the world wherever plants of the mustard family grow. In rare instances, it may become of economic importance in the field, greenhouse, or seedbeds, especially in radish seedbeds. At times it is important in mustard. The disease is of no importance on the market except on radishes marketed with the tops and on mustard greens.

The fungus may attack any or all of the plant parts above ground. The leaves, stems, and fruiting organs may become thickened and distorted. Just underneath the surface of the stems or leaves great numbers of milky-white spores are borne in chains. When a large quantity of these spores form under the epidermis, the tissues die and may split open, thus making visible the white, powderlike spores. It is these masses of spores, appearing as white pimples or ridges, which

have given rise to the name white rust. The lesions have yellow borders, which set them off sharply from the healthy green tissues. Decided swellings, with resultant malformation, often occur in fleshy tissues. Young plants and healthy, vigorous plants usually are more subject to attack than older ones or those weakened by unfavorable conditions.

CAUSAL FACTORS

The causal fungus, *Albugo candida*, overwinters on plant debris and probably in the perennial parts of affected plants. In the spring it produces spores that are scattered by wind and water. The amount of infection produced is determined by temperature and moisture conditions. The spores germinate at temperatures almost down to 32° F. and as high as 77°, but do best at about 50°. Under favorable conditions they will germinate within 2 hours. In the field the spores germinate mostly in the morning, when it is cool and when there is dew on the plants to provide the necessary moisture. Hot, dry weather arrests the progress of the disease.

CONTROL MEASURES

Affected plants, both cultivated and wild, should be destroyed. The soil should be kept only moderately moist. Spraying with 4–4–50 bordeaux mixture will prevent the appearance of the disease if it is done early and often enough to keep the leaf surfaces covered (*51*, *55*).

RUTABAGA AND TURNIP

The turnip of the market is the fleshy taproot and hypocotyl of the turnip plant (*Brassica rapa*), a member of the mustard family. The rutabaga is *Brassica napo-brassica*. Various horticultural varieties are grown, but in all of them tenderness, crispness, and freedom from blemishes and decay are the important factors in their marketability. Young turnips are marketed with the tops attached as a bunch vegetable; the late crop and rutabagas are topped and stored like other root vegetables. In the former, consequently, good color of the leaves and freedom from blemishes and decay are direct market factors, but in the latter these are of only indirect significance.

Turnip and rutabaga are subject to several leaf diseases, some of which also cause root rots. These are alternaria leaf spot (p. 4), alternaria root rot and leaf spot (*A. herculea* (Ell. and Martin) J. A. Elliott), anthracnose (*Colletotrichum higginsianum* Sacc.), downy mildew (p. 13, and pl. 9, *B*), powdery mildew (*Erysiphe polygoni* DC.), mosaic (p. 17), white rust (p. 33), and white spot (*Cercosporella albomaculans* (Ell. and Ev.) Sacc.).

The root is affected with bacterial soft rot (p. 5), black rot (p. 10), club root (p. 12), gray mold rot (p. 17), rhizoctonia rot (p. 18), watery soft rot (p. 21), and yellow (p. 23). Bacterial soft rot is especially severe in young turnips when they are shipped with the tops attached.

ALTERNARIA ROOT ROT AND LEAF SPOT

(*Alternaria herculea* (Ell. and Martin) J. A. Elliott)

The leaf spots of crucifers caused by various species of *Alternaria* are in general very much alike (p. 4). *A. herculea*, however, has

been found to cause more or less serious decay of turnip roots in the field and in storage pits when seasonal conditions are favorable. During warm, moist weather the leaves are sometimes so severely affected that death of the plant results; in other instances the plants are injured only enough to cause stunted and unmarketable roots. When the tops are seriously affected there is danger of loss of the root crop during storage and marketing. The fungus may enter the root through the crown, or infection may take place when spores and mycelium come in contact with uninjured surfaces of the root. Bruised and scarred roots are especially susceptible to infection.

Lesions produced on the roots are usually circular, dark brown, and firm. Frequently concentric rings of light and dark-brown tissue give them a characteristic appearance. Gray to grayish-brown surface mycelium may form over the decayed areas in humid storages or pits. Although the dark-brown decay produced may penetrate deeply into the roots, it remains firm and is not moist nor ill-smelling as in roots affected with bacterial soft rot.

Control of this disease in the field has not been worked out, although preliminary experiments indicate that when the seed is known to be infected a hot-water treatment (122° F. for 10 minutes) may be of value.

Turnips intended for storage in pits or warehouses should be as free from wounds and bruises as possible and should be carefully selected for freedom from decay (*9*, *82*).

ANTHRACNOSE

(*Colletotrichum higginsianum* Sacc.)

This disease occurs on many crucifers, but is seldom of economic importance. Turnips, radishes, cabbage, Chinese cabbage, and collards are the most susceptible crops. The stems and leaves are most generally attacked, although the seed pods and roots are sometimes affected. Grayish-tan circular spots are produced on the leaves, and elongated lesions are caused on the stem. In humid weather very small pink spore masses cover the surfaces of the larger lesions. In very severe attacks, the plants are so weakened that the marketability of the crop is greatly reduced.

In stored turnips, this fungus sometimes causes grayish to light tan spots with regular, depressed margins. Bacterial soft rot usually follows in these spots and a light-brown wet decay encircles the original lesion. Under humid conditions the spores and mycelium are able to cause infection of uninjured turnip roots, consequently this fungus and the associated bacteria common to most soils are capable of causing considerable damage to storage turnips under favorable moisture and temperature conditions.

No control measures have been developed (*9*, *13*, *31*).

BROWN HEART

In parts of New England and Canada there is sometimes a serious discoloration of the roots of rutabagas that appears to be due to environmental factors. Irregular streaks and patches of discolored parenchyma tissue and water-soaked areas have led to the common names "water core," "mottled heart," "dark center," and "brown

heart." Frequently the marketable roots are so badly discolored that they are worthless. No organisms of any kind have been found associated with this trouble. A chemical analysis shows that the affected tissues are low in sugars and that there is an increase in proteins, fiber, and ash over that normally found in healthy stock.

It has been reported that in New Brunswick, Canada, brown heart of turnips was prevented by application of sodium metaborate to the soil at the rate of 10 pounds per acre. The application of 15 to 20 pounds of borax per acre to the soil in Massachusetts has controlled this disease (*32*, *49*).

WHITE SPOT

(*Cercosporella albomaculans* (Ell. and Ev.) Sacc.)

The leaves of turnips, radishes, and Chinese cabbage are sometimes injured by a leaf disease called white spot. In most instances the spotting is not severe enough to affect the development of the plants, but the marketability of the leaves for use as greens is greatly reduced. First signs of infection are small water-soaked spots on the leaf blades. As the tissue is killed it becomes bleached ashen gray to white. Most spots are one-eighth to one-fourth inch in diameter and circular in outline. When the spots become numerous they may coalesce and cause yellow and brown areas of larger size and induce a premature yellowing of the whole leaf.

The causal fungus apparently lives over from season to season in diseased plant refuse left in the field, but usually it causes so little damage that development of control measures has not been necessary (*12*, *21*).

CUCURBITS

The cucurbits belong to what is known as the gourd family, which includes a large number of wild species, the various ornamental gourds, and the following important food plants: Cucumber (*Cucumis sativus* L.); muskmelon (*C. melo* L.); pumpkin and squash (*Cucurbita pepo* L., *C. moschata* Dcne., and *C. maxima* Dcne.); and watermelon (*Citrullus vulgaris* Schrad.).

The following literature citations refer to publications each of which deals either with several diseases on a given cucurbit crop or with the diseases of several of the cucurbits, particularly with reference to the control of these diseases: *99*, *148*, *161*, *166*, *169*, *170*, *171*, *195*, *196*, *197*, *201*, *203*, *208*.

CUCUMBER

Cucumbers are harvested while green and their desirability depends chiefly upon their crispness, firmness, shape, and color. The size and shape of the cucumber fruits vary greatly with different varieties. For fancy pickles, the smaller varieties are grown and the fruits are harvested when they reach a length of 1 or 2 inches. The larger kinds are grown for slicing, in salads, and for keg or barrel pickles. There is very little market for ripe cucumbers. In general, any white or yellowish fruits are looked upon with disfavor, although in recent years white pickles have been placed upon the market with success.

The cucumber plant is susceptible to many diseases that greatly influence the quantity and the quality of the fruits. To produce well-colored, well-shaped, and firm fruits of normal size, the vine must not only be free from disease but it must be in vigorous growing condition. Fruits on diseased or impoverished vines do not grow to normal size; they are poorly shaped, the blossom-end often being pointed, crooked, or flabby; they develop poor color, or yellow prematurely; and they wilt or shrivel soon after picking.

Several cucumber diseases are not characterized by lesions on the fruits, but they seriously affect the vines, and thus indirectly the fruits. These are: Bacterial wilt (*Bacillus tracheiphilus* E. F. Smith[4]), downy mildew (*Peronoplasmopara cubensis* (Berk. and Curt.) Clint.), and root knot (*Heterodera marioni* (Cornu) Goodey). Less important are fusarium wilt (*Fusarium* sp., p. 60), the leaf spots and blights (*Cercospora citrullina* Cke., *Macrosporium cucumerinum* Ell. and Ev., *Phyllosticta cucurbitacearum* Sacc., *Septoria cucurbitacearum* Sacc., and *Stemphylium cucurbitacearum* Osner), powdery mildew (*Erysiphe cichoracearum* DC.), and verticillium wilt (*Verticillium albo-atrum* Reinke and Berth.).

Other cucumber diseases occur on both the vines and the fruit and thus are of direct and indirect effect in the market. The most important diseases of this type are anthracnose (*Colletotrichum lagenarium* (Pass.) Ell. and Mals.[5]), bacterial spot (angular leaf spot) (*Bacterium lachrymans* E. F. Smith and M. K. Bryan),[6] cottony leak (*Pythium aphanidermatum* (H. A. Edson) Fitz.), mycosphaerella black rot (*Mycosphaerella citrullina* (C. O. Smith) Gross.), scab (*Cladosporium cucumerinum* Ell. and Arth.), sclerotinia fruit and stem rot (*Sclerotinia sclerotiorum* (Lib.) DBy.), sclerotium rot and blight (*Sclerotium rolfsii* Sacc.), and the virus disease, mosaic.

A number of diseases occur only on the fruits. These are bacterial soft rot (*Bacillus* spp.), blue mold rot (*Penicillium* spp.), diplodia rot (*Diplodia* sp., p. 62), fusarium rot (*Fusarium* spp.), gray mold rot (*Botrytis* sp.), pink mold rot (*Cephalothecium roseum* Cda.), rhizopus soft rot (*Rhizopus* spp.), low temperature break-down, and sunscald.

ANTHRACNOSE

(*Colletotrichum lagenarium* (Pass.) Ell. and Hals.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Anthracnose is most serious as a disease of melons, but also occurs on cucumbers. It affects the vines, causing reduced yields due to foliage and stem injury, and malformed fruit. It also occurs on fruits in the field and in transit, disfiguring them and causing decay, either directly or by opening the way for secondary invaders. The disease, especially in its direct effect upon fruits, is most severe in those cucurbit crops, such as watermelons, whose fruits are long exposed to infection in the field.

In the cucumber, losses are most severe when the field crop is grown for slicing purposes, or on cucumbers grown under glass.

[4] Syn., *Erwinia tracheiphila* (E. F. Smith) Winslow et al., 1920.
[5] Syn., *Glomerella lagenarium* (Pass.) Stevens (*187*).
[6] Syn., *Phytomonas lachrymans* (Smith and Bryan) Bergey et al.

Damage also tends to be heavy in fruits that are shipped long distances because of the additional time available for the development of lesions in transit.

On leaves, the lesions generally appear first on veins and become angular in shape because of the more rapid development along the veins. The lesions consist of reddish-brown, dry, dead tissues, which in the early stages often are surrounded by a narrow, yellowish, translucent, or water-soaked border. As the lesions enlarge they become more circular and blotchlike.

On fruits the first symptoms are the appearance of more or less circular sunken, water-soaked areas. As the lesions enlarge, the fungus sporulates abundantly, producing orange-pink slimy spore heaps or a slimy pink coating (pl. 10, *E*, *B*); later the lesions turn black. On mature fruits the black lesions may show white centers on which old acervuli appear as conspicuous black dots. The tissues underneath are dry and the sunken epidermis often cracks, exposing the tissues to invasion by soft rot organisms.

CAUSAL FACTORS

The causal fungus, *Colletotrichum lagenarium*, overwinters in plant debris of the previous season and on seed from diseased fruit. The disease usually starts in isolated centers consisting of one or two infected plants, which have contracted the disease from the soil or from the seed. The fungus spreads from these centers and increases in abundance, but usually the disease becomes general in a field late in the season.

Since the spores are sticky, the principal agencies of dissemination in the field are insects, rain, and surface drainage water. During rains the spores are washed to the soil and then splashed to healthy leaves. The spores are likewise readily spread by splashing water from overhead irrigation systems. They are also disseminated during cultural operations.

The most favorable temperatures for spore germination lie between 71° and 80° F. The spores will not germinate when the temperature is either below 40° or above 86°; furthermore, penetration of the tissues by the fungus does not take place when the temperature is lower than 50°. The incubation period is about 5 days under the most favorable temperature and moisture conditions; consequently, fruits harvested from diseased vines often appear sound but develop lesions in transit or on the market. Since the fungus can penetrate the rind of mature fruit, new lesions develop as long as temperature and moisture conditions are favorable for spore germination.

CONTROL MEASURES

Crop rotation should be practiced because the fungus can overwinter in the soil on plant debris of the preceding year. No field should be planted successively to cucurbit crops.

In view of the fact that infected seed often serves as a source of inoculum, it is urged that only seed from fruit free from anthracnose be used. If disease-free seed cannot be secured, the seed should be disinfected by soaking for 5 minutes in a solution of mercuric chloride (1 part to 1,000 parts of water), after which it should be washed thoroughly in running water and dried.

A, Low temperature break-down of cucumber followed by various fungi; *B*, anthracnose fruit spot of cucumber; *C*, mosaic disease of cucumbers.

A, Bacterial spot of cucumber; *B*, cladosporium fruit spot or scab of cucumber.

Spraying with bordeaux mixture, using as strong a solution as is permissible without causing injury, is an effective control measure. The spraying should be thoroughly done and power sprayers should be used so that both sides of the leaves will be covered. For details consult local county agent or State or Federal plant pathologists.

Considerable progress has been made in the development of resistant varieties, particularly of watermelons. (See Watermelon, Anthracnose, p. 58) (*121*, *125*, *126*, *152*, *159*, *166*, *169*, *183*, *187*.)

BACTERIAL SOFT ROT

(See Cabbage, Bacterial Soft Rot, p. 5; Cucumber, Bacterial Spot, p. 39; and Muskmelon, Bacterial Soft Rot, p. 48.)

BACTERIAL SPOT (ANGULAR LEAF SPOT)

(*Bacterium lachrymans* E. F. Smith and M. K. Bryan)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Bacterial spot of cucumber occurs on both the leaves and the fruits. The name "angular leaf spot" has been applied to the disease as it appears on the leaves. None of the horticultural varieties of cucumber now grown is known to be resistant to this disease. It does not occur on the watermelon, muskmelon, or squash. Reduction of leaf surface brought about by severe infection often leads to a reduction in yield, and, so far as the pickle industry is concerned, losses are principally of this nature, although considerable loss may result from rotting of small fruits on the vines. When cucumbers are grown for slicing purposes, however, opportunity is given for development of the lesions on grown fruit.

The fruit lesions, which are at first evident as minute, circular, water-soaked areas, later become conspicuous because the centers sink and take on a chalky-white color as a result of the drying out and cracking of the affected tissues (pl. 11, *A*). Often a gummy exudate is formed on the lesions before the spots become dry. The lesions are usually blemishes confined to the outer tissues of the fruits. Frequently, however, these symptoms are followed by a rapid breakdown of the infected fruits with an extensive soft rot that occurs particularly during transit and marketing periods. Most investigators consider this to be the result of invasion of the bacterial spot lesions by secondary soft-rot-producing bacteria. Others have obtained evidence suggesting that, under certain conditions, the organism causing angular leaf spot and bacterial spot of the fruit can also cause an extensive soft rot of the fruits.

The first manifestation of the disease on the cotyledons (seed leaves) and the true leaves is the appearance of water-soaked spots. On the cotyledons these are usually round, while those formed on the leaves extend along the veins and become distinctly angular in shape. Under moist conditions the spots may increase in number until the entire leaf is covered.

Early in the morning tearlike drops of exudate swarming with bacteria are often suspended from the lower surface of such spots. About a week after the leaf spots are noticeable, they become dry and white to tawny in color. Rain often knocks out these dry spots,

thus giving the leaves a very ragged appearance. When the stems or leaf petioles become affected they are water-soaked, and where bacterial exudate dries over their surface a white incrustation is produced. When weather conditions are favorable for the development of the disease early in the season, the young vines are often badly stunted. The leaf surface is reduced and this eventually causes appreciable reduction in yield.

CAUSAL FACTORS

The disease is caused by a bacterial organism, *Bacterium lachrymans*. The bacterium is carried on the seeds and consequently seedling infection is the first evidence of the disease in the field.

The first lesions appear on the cotyledons and later the angular spots appear on the first true leaves. The bacteria enter the tissues through the stomata. It happens occasionally that seedlings from healthy seed are infected from the soil, and from one or more leaf spots on a few seedlings the disease may spread over an entire field and be carried to neighboring fields.

The development of the causal bacterium is checked by dry, hot weather. Rainfall, particularly if accompanied by wind, spreads the disease because bacteria are spattered by raindrops from diseased plants and soil to healthy plants. Infested soil blown by the wind also disseminates the bacteria. Surface drainage water, overhead sprinkling water, and the hands and clothes of pickers also act as transfer agents.

Inoculum may get onto fruit in the field or during the packing process. In view of the fact that at least 6 days are required for the development of visible lesions on fruit after artificial inoculation, it is possible that fruits showing young lesions upon arrival at the market may have appeared sound when picked and packed.

CONTROL MEASURES

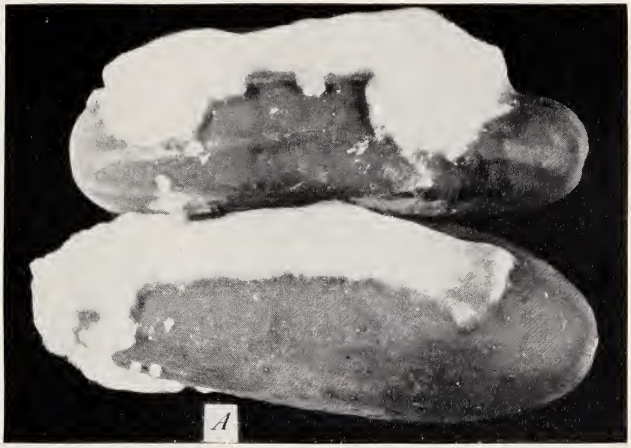
The most effective control program is a combination of seed disinfection and crop rotation. The seed should be immersed for 5 minutes in a solution of mercuric chloride (1 part to 1,000 parts of water) and then washed thoroughly in water and dried.

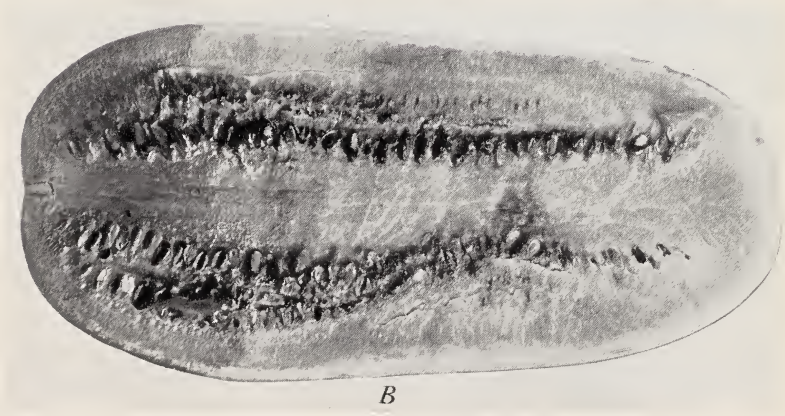
Soil that has not previously been planted to cucumbers should be used; if it is not available, at least 2 years should be allowed to elapse before replanting to cucumbers. If the disease is present in a field, it is advisable not to work among the vines early in the morning or while the foliage is wet with rain. When spotted fruit must be shipped or stored it should be packed dry and kept in a cool, well-ventilated place (*65, v. 2, pp. 209–299; 95; 96; 98; 130; 149; 165; 186; 202*).

BACTERIAL WILT

(*Bacillus tracheiphilus* E. F. Smith)

Bacterial wilt is one of the most serious vine diseases of cucurbits. It occurs on cucumber, muskmelon, summer and winter squash, pumpkin, and rarely on watermelon. The fruit of diseased vines is usually small, poorly shaped, and of poor flavor.





A, Cottony leak of cucumber; *B*, pythium rot of watermelon.

The disease occurs in practically all States where cucumbers are grown. The causal bacterium, *Bacillus tracheiphilus*, overwinters in two insects, the striped and the 12-spotted cucumber beetles. Insects contaminated with the bacteria feed on the young plants in the spring and thus initiate the disease in the field. Other beetles become infected while feeding on the diseased vines and thus help in spreading the disease. The causal organism is never disseminated by either the soil or the seed.

The causal agent can enter the plant only through wounds. Since infection proceeds from bites made by the insects lesions may appear anywhere on the above-ground parts of plants. Once the bacteria get into the water-conducting tissues, progressive wilting of the leaves and stems proceeds in either direction from the point where the insect has fed. If the central stem is infected the whole vine usually wilts rapidly. If such a stem is cut, a white viscid exudate oozes out of the water vessels and when touched with the finger can be drawn out into long very thin threads. This characteristic serves to distinguish bacterial wilt of cucurbits from that caused by *Fusarium* sp.

Climatic factors are significant chiefly as they affect the abundance and activities of the beetles. The disease, however, is usually more severe under conditions that produce succulent growth.

Control of the disease involves control of the beetles. These avoid plants that are covered with spray. For this reason spraying with bordeaux mixture with 1½ pounds of lead arsenate per 50 gallons as a stomach poison for those beetles that do not avoid the leaves is recommended. Various dusts are also effective. Diseased vines should be pulled as soon as seen, and destroyed to remove the source of inoculum. The use of an early catch crop of squash scattered around the edges of the field has been recommended (*65, v. 2, pp. 209–299; 101; 103; 110; 111; 179; 180; 181; 182*).

COTTONY LEAK

(*Pythium aphanidermatum* (Eds.) Fitz.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

So far cottony leak has been reported from only a few localities in the Southeastern States. Infection of cucumber fruits takes place in the field, and decay develops and spreads very rapidly during transit. The fungus is spread by contact, and visible wounds are apparently not necessary to enable it to penetrate the fruits. The fungus also causes damping-off and a vine canker during excessively wet seasons.

The first symptoms on the fruits are soft, dark green, water-soaked lesions; later, as the mycelium penetrates the tissues of the fruit, water is liberated in large quantities. In the moderately humid atmosphere of containers during transit, a very luxuriant, white, cottony mycelium is produced, which sometimes completely covers the fruit (pl. 12, *A*). When juice from decaying cucumbers leaks down upon those below, the mycelium becomes flattened and forms a matted layer over the surfaces of the fruits. Unless there are other contaminating organisms present, the cucumbers will decay completely without disagreeable odor.

CAUSAL FACTORS

The causal fungus, *Pythium aphanidermatum*, is a soil inhabitant. It causes greatest damage in wet soils and in seedbeds where the plants are closely crowded together. This fungus produces no aerial spores, but depends upon propagation by mycelium and by spores that swim about in water during the rainy season. Plants or fruits that come in contact with the infected soil or that become spattered with rain and soil containing spores or mycelium of *Pythium* are consequently liable to infection.

CONTROL MEASURES

This disease may be controlled in ordinary seasons by providing good drainage, and by thinning out the plants. Cucumbers showing any evidence of decay at time of harvest should not be shipped (*118*, *120*).

DOWNY MILDEW

(*Peronoplasmopara cubensis* (Berk. and Curt.) Clint.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

During certain seasons downy mildew is a serious field disease of cucumber and muskmelon, particularly in the Atlantic and Gulf coast regions. It is likewise of importance on greenhouse cucumbers. The disease occurs on the vines, often causing premature death. Badly affected plants set few fruits. The disease is thus a market factor even though no lesions are produced on the fruits. Cucumbers that do set, grow slowly and become misshapen. Muskmelons from diseased vines are frequently of poor eating quality. The pumpkin and squash are susceptible, but the disease is seldom serious on them. The watermelon seldom, if ever, is attacked.

The disease is recognized by the presence of yellowish, rather indefinitely delineated spots formed on the older leaves. Under humid conditions a fine downy mildew covered with a faintly purplish powder, the spores of the fungus, can be observed on the lower side of the leaf spots. Old spots become papery and light brown in color. During favorable conditions the spots may enlarge until the entire leaf is affected. Severely diseased plants grow poorly and die prematurely.

CAUSAL FACTORS

The causal fungus, *Peronoplasmopara cubensis*, is one of the downy mildews. It does not overwinter in plant debris or in the soil, nor is it carried on the seed. All evidence indicates that the organism overwinters on growing cucurbits in the Gulf States and that the spores are carried by winds to more northerly plantings in the spring. In favorable seasons the disease thus advances northward progressively from section to section during the spring and summer until it reaches the northern growing areas. The disease is favored by rainy periods during which the nights are cool and the days comparatively warm.

CONTROL MEASURES

The disease can be controlled by the use of bordeaux sprays or copper-lime dusts. Repeated applications of the fungicide at fre-

quent intervals are necessary and the leaves must be thoroughly covered (*103*, *104*, *116*, *188*, *197*, *204*).

FUSARIUM ROT

(See Muskmelon, Fusarium rot, p. 51.)

LOW TEMPERATURE BREAK-DOWN

Cucumbers held for 1 week or longer at temperatures of 32° to 40° F. frequently show low temperature break-down soon after they are removed from storage. This injury is recognized by the presence of numerous sunken spots over the surface of the fruits (pl. 10, *A*), the total area of which may cover half to three-quarters of the cucumber surface. Frequently the tissues immediately at the base of the spines are first affected; likewise those about the lenticels appear to be involved in most cases. The sunken areas are soon spotted with molds and affected with shallow decays caused by species of *Fusarium* and other fungi.

Little is known regarding the exact conditions necessary to produce this injury. In hitherto unreported studies [7] cucumbers were held at different storage temperatures for 1 week, following which they were placed at 60° F. for 4 to 5 days. Severe injury occurred at 32°, slight injury at 40°, and no injury at 50°.

MOSAIC

OCCURRENCE, SYMPTOMS, AND EFFECTS

Mosaic is one of the most serious diseases of cucumber and is likewise of importance on muskmelon, squash, and pumpkin. Its occurrence on watermelon has been observed on several occasions. On cucumber it causes losses in greenhouses as well as in the field. It affects the vines, which are stunted and killed, thus causing reduced yields of fruit. It also affects the fruit directly, causing mottling and suppression of chlorophyll development (white pickle), development of warts, distortion, and stunting (pl. 10, *C*).

The symptoms appear first as patches of yellowish or lighter green in the dark green portions of the young leaves. The latter are thicker than the light areas and somewhat raised, so that the leaves appear crinkled and blistered and even curled at the top, or more often along one side, giving a characteristic drooping of one side of the leaf. Older leaves also curl and turn yellow. The yellowing and death at first extend as wedge-shaped areas from the margin inward along the veins but eventually involve the entire leaf, which withers. Affected plants may live in the field in a stunted condition until killed by frosts, but in greenhouses they may wither and die within 15 to 20 days. When young plants are affected their growth usually is checked at once; they remain stunted and do not set fruit.

On muskmelon the leaf symptoms are similar to those on cucumber, except that the darker spots are more prominent, infected leaves show a more conspicuous curling, and although they become yellowed they do not tend to die prematurely. The fruits show little evidence of the disease aside from a reduction in size and poor quality of flesh.

[7] WRIGHT, R. C. Data on file in Bureau of Plant Industry.

On the young leaves of squash and the pumpkin the lighter colored yellow areas tend to coalesce and form large blotches while the darker green areas are prominently raised above the rest of the leaf surface. The fruits are less affected than are those of the cucumber although some wartiness does occur.

CAUSAL FACTORS

Cucumber mosaic is one of the virus diseases. Although this disease is very infectious it is not caused by bacteria, fungi, or other organisms, such as are responsible for the production of other plant diseases. The infective principle is spoken of as a virus. It can be transmitted by any method that involves the transfer of the plant juice from a diseased plant to a healthy one. This may be accomplished by crushing a portion of a leaf from an infected plant and rubbing it onto an injured surface of a healthy leaf. The juice remains infectious even after it is diluted with water to 1:10,000.

Although the cucumber mosaic virus is readily transmitted through the seed of the wild cucumber (*Micrampelis lobata* (Michx.) Greene) it is believed that it is rarely, if ever, transmitted through the cultivated cucumber seed. However, it has recently been shown that seed transmission occurs much more frequently in the muskmelon than was formerly believed. The virus overwinters chiefly in the seed of the wild cucumber and in the roots of the following perennial plants: Milkweed (*Asclepias syriaca* L.), pokeweed (*Phytolacca americana* L.), catnip (*Nepeta cataria* L.), and certain species of the groundcherry (*Physalis* spp.). When the leaves of these infected plants appear in the spring they are fed upon by various insects that later feed on healthy cucumber plants, thus introducing the virus into the new crop. Similarly, they spread the virus through the field during the growing season. The insects prominently involved are the melon aphid (*Aphis gossypii* Glover), the striped cucumber beetle (*Diabrotica vittata* Fabr.), and the 12-spotted cucumber beetle (*D. duodecimpunctata* Fabr.), but the melon aphid is the most important disseminating agent.

CONTROL MEASURES

Mosaic usually can be controlled in the greenhouse crop by fumigating regularly and often for the control of the insect carriers. All mosaic-diseased plants should be cut off at the base as soon as they are discovered. It is not advisable to remove the cut plants because of danger of spreading infection.

Control in the field is only partially successful. The most important measures are removal of all weed hosts in the field and for a radius of 50 to 75 yards from the borders of the field and control of the transmitting insects by spraying or dusting. Some progress has been made in the development of resistant varieties (*88*, *100*, *102*, *103*, *108*, *109*, *112*, *113*, *114*, *115*, *128*, *139*, *140*, *142*, *143*, *144*, *151*, *160*, *176*, *177*, *178*, *198*, *199*, *200*).

MYCOSPHAERELLA BLACK ROT (MYCOSPHAERELLA WILT; GUMMY STEM BLIGHT)

(*Mycosphaerella citrullina* (C. O. Smith) Gross.)

Mycosphaerella black rot, caused by the fungus *Mycosphaerella citrullina*, has been found an important cause of decay in cucumber

shipments from Florida. The decay appears first in the form of water-soaked lesions that later turn dark. The affected tissues are firm, due to the compact mycelial development of the causal organism immediately beneath the epidermis. Experimental lots of cucumbers inoculated in Florida were badly decayed upon arrival at New York City. The fruiting bodies of the fungus (both pycnidia and perithecia) were found on many of the cucumbers. Although the decay may continue to develop during transit there are no indications that new lesions appear after the cucumbers are harvested (*164*).

(See Watermelon, Mycosphaerella Black Rot, p. 60.)

PINK MOLD ROT

(See Muskmelon, Pink Mold Rot, p. 54.)

POWDERY MILDEW

See (Muskmelon, Powdery Mildew, p. 54.)

RHIZOPUS SOFT ROT

(See Muskmelon, Rhizopus Soft Rot, p. 55.)

SCAB

(*Cladosporium cucumerinum* Ell. and Arth.)

Scab is an important disease of cucumbers, and likewise occurs on pumpkin, muskmelon, and squash. On cucumbers it is of chief importance on the greenhouse crop and on the pickle crop in the North Central States.

The cause of the disease is the fungus *Cladosporium cucumerinum*. It overwinters on plant debris in the soil. The cotyledons, young leaves, and stems are first involved. Black rounded spots appear on the former and longitudinal black stripes on the latter. Older leaves are later involved to some extent, but it is on the fruits that the chief injury occurs. Small, grayish, slightly sunken spots mark the first appearance of the disease there. Frequently a gummy exudate forms on the lesions which later turns dark. Under dry conditions the lesions may remain as more or less superficial blemishes; under humid conditions, however, the fungus causes a shallow decay. In later stages the decayed tissues become covered with a dark olive-green layer composed of the mycelium and spores of the causal fungus.

The disease is favored by cool, moist weather inasmuch as the pathogen grows best at about 70° F., and develops only slowly at 95°. It is particularly severe under humid conditions. Lesions apparent at the time of shipping may continue to develop during transit, but there is no development of new lesions.

This disease can be controlled to some extent in the greenhouse by sanitary practices and by disinfection of the soil. Spraying the field crop with bordeaux mixture appears to have been successful under certain conditions. Little progress has been made in the development of resistant varieties. In transit, further development on affected fruit can be retarded by keeping it dry (*89*, *90*, *97*, *107*, *129*, *141*, *168*, *172*).

WATERY SOFT ROT

(See Cabbage, Watery Soft Rot, p. 21.)

MUSKMELON

The muskmelon, *Cucumis melo* L., is one of the many species belonging to the gourd family, the Cucurbitaceae. There are several botanical varieties within the species. Although some of the forms are not actually musk-scented, all members of the species are referred to as muskmelons. The term "cantaloup," in a strict botanical sense, refers only to a group of hard scaly-rinded muskmelons, few of which are grown in the United States. Commercially it is used synonymously with muskmelon. Although not botanically correct, the term will be used in this publication to denote the small green-skinned, netted melons that are the most important single type of muskmelon found on the American market. Other types of muskmelon in the approximate order of their importance are Honey Dew melon, Honey Ball melon, Persian melon, Casaba melon, Spanish melon, and Santa Claus melon.

The market value of these fruits depends upon their flavor and freedom from blemishes and decay. The flavor depends primarily upon the variety and the stage of maturity at which the fruit is harvested. In order to reach the market in an acceptable condition the melons must be picked while still firm, yet if they are removed from the vine too soon they lack flavor.

The muskmelon plant is subject to many field diseases with direct or indirect effects on the fruit. Such diseases as bacterial wilt (p. 40), fusarium wilt (p. 60), root knot, mycosphaerella wilt (p. 44), and thielaviopsis root rot (*Thielaviopsis basicola* (Berk.) Ferraris) are characterized by root or vascular troubles, which result in the stunting and wilting of the vines and a poor setting and poor quality of fruit. Cercospora leaf spot (*Cercospora citrullina* Cke.), downy mildew (p. 42), macrosporium leaf blight (*Macrosporium cucumerinum* Ell. and Ev.), powdery mildew, septoria leaf spot (*Septoria cucurbitacearum* Sacc.), and the virus disease, mosaic (p. 43), affect the foliage and thus curtail production. With anthracnose, watery soft rot (p. 21), and sclerotium rot both the vines and the fruit in the field are involved.

Muskmelon fruits are subject to the following decays during the transit and marketing periods: Alternaria rot (*Alternaria* spp.), anthracnose, bacterial soft rot, blue mold rot, charcoal rot (*Rhizoctonia bataticola* (Taub.) Butler),[8] cladosporium rot (*Cladosporium cucumerinum* Ell. and Arth.), diplodia rot (*Diplodia natalensis* Ev.), fusarium rot, phytophthora rot (*Phytophthora* sp.), pink mold rot, and rhizopus soft rot (*Rhizopus* spp.).

Melons affected with sunburn and with low temperature breakdown are also found on the market.

ALTERNARIA ROT

(*Alternaria* spp.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Alternaria rot is commonly found on the market on all types of muskmelons. It is characterized by a mold and a shallow decay at the stem scar of cantaloups and Honey Ball melons but may occur

[8] Syn., *Macrophomina phaseoli* (Maubl.) Ashby.

Various decays following low temperature break-down of Honey Dew melon: Large black area, alternaria rot; small scattered black spots, cladosporium rot; greenish blue spots, blue mold rot; red spots, fusarium rot.

in the form of definite lesions anywhere over the surface. On all white-skinned varieties the conspicuous spots disfigure the fruit and open the way for other rot-producing organisms. The decay often follows sunscald and low temperature break-down (pl. 13).

Alternaria rot first appears on Honey Ball and Honey Dew melons as small brown spots that later enlarge to form oval lesions varying from less than an inch to several inches in diameter (pl. 14, *A*). Large areas of the melon are frequently covered by a single lesion formed by the enlarging and coalescing of numerous small ones. The lesions are russet or chestnut brown in color, frequently with alternating bands of different shades forming irregular concentric rings. The decayed tissues may form a thin papery layer largely confined to the rind, or they may extend a half inch into the flesh forming a grayish-black moist spongy mass that can be easily lifted out of the healthy flesh. Later a gray, greenish-gray, or black matted growth of mold may develop over the surface, or the lesion may become covered with a shallow, black, velvety layer in which the spores of the causal fungus are abundantly produced.

The symptoms on cantaloup and Persian and Casaba melons are similar to those described above, except that the external injury is less conspicuous, with little brown discoloration, so that diagnosis of the decay is largely dependent upon the mycelium and spore development of the causal organism.

CAUSAL FACTORS

Studies on the New York market have shown that most of this decay is caused by species of *Alternaria* distinct from the organism responsible for the leaf blight of muskmelon and other cucurbits. (See Macrosporium Leaf Blight, p. 52.)

The development of the causal organism in culture and the decay of melons is most rapid at temperatures of 75° to 85° F., and slow between 35° and 45° F.

CONTROL MEASURES

Little is known regarding the conditions that favor infection of melons by these fungi. It appears that infection is favored by the presence of wounds and that it may occur at any time following harvest.

The fact that the causal organisms develop only slowly at low temperatures indicates that the disease can be fairly well controlled in transit by proper refrigeration. Prompt handling of melons picked in sound condition and well refrigerated in transit should prevent any significant loss from the disease (*123*, *158*, *206*).

ANTHRACNOSE

(*Colletotrichum lagenarium* (Pass.) Ell. and Hals.)

Anthracnose is a serious field disease of muskmelons and other cucurbits. On the muskmelon the disease is recognized by the presence of spots on the leaf veins, leaf blades, petioles, runners, fruit pedicels, and fruit. All spots other than those on the leaves and fruit are long and narrow. On the leaves they are at first yellow or water-soaked. As they enlarge they turn brown in the center and become angular.

Lesions on the fruit are at first dark green or water-soaked, later turning reddish brown. As they enlarge they become round to oval and definitely sunken. Infected melons are frequently rotted by secondary organisms which enter at the anthracnose injuries. Melons from diseased vines are usually poorly developed and are flat or insipid in flavor. The flesh of infected melons, particularly near the lesions, has a bitter taste. Lesions on all parts of the plant become covered with the pink spore masses which are formed in abundance, particularly during periods of wet weather.

Within recent years anthracnose has ceased to be of great importance on the market. It practically never is found on western muskmelons of any type and only seldom on eastern-grown cantaloups. It is, however, occasionally found on eastern-grown Honey Dew melons (*158*, *206*).

(See Cucumber, Anthracnose, p. 37, for causal factors, control measures, and further literature citations.)

BACTERIAL SOFT ROT

(*Bacillus* spp.)

Bacterial soft rot has been reported as an important fruit rot of eastern-grown cantaloups in the field during certain seasons. It is only of minor importance on the market on domestic muskmelons. However, the disease occasionally causes considerable loss in Honey Dew melons imported from South America. Infected melons are soft and mushy and eventually become hollow shells filled with the nearly liquefied diseased tissue. Later the melons collapse and spill the contents over adjacent ones and over the packing material so that the crates become wet and leaky.

An organism described as *Bacillus melonis* Gidd. was at one time found to cause the decay of cantaloups in the field. Cucumbers were likewise rotted when artificially inoculated with the same bacterium. This organism differs so little from the common soft rot pathogen, *B. carotovorus* L. R. Jones, that it probably should be considered as belonging to the latter species. No studies have been made to determine the taxonomic position of the organism causing the decay of Honey Dew melons on the market. It is probably closely related to the group of soft rot organisms represented by *B. carotovorus* (*65*, *99*, *127*, *206*).

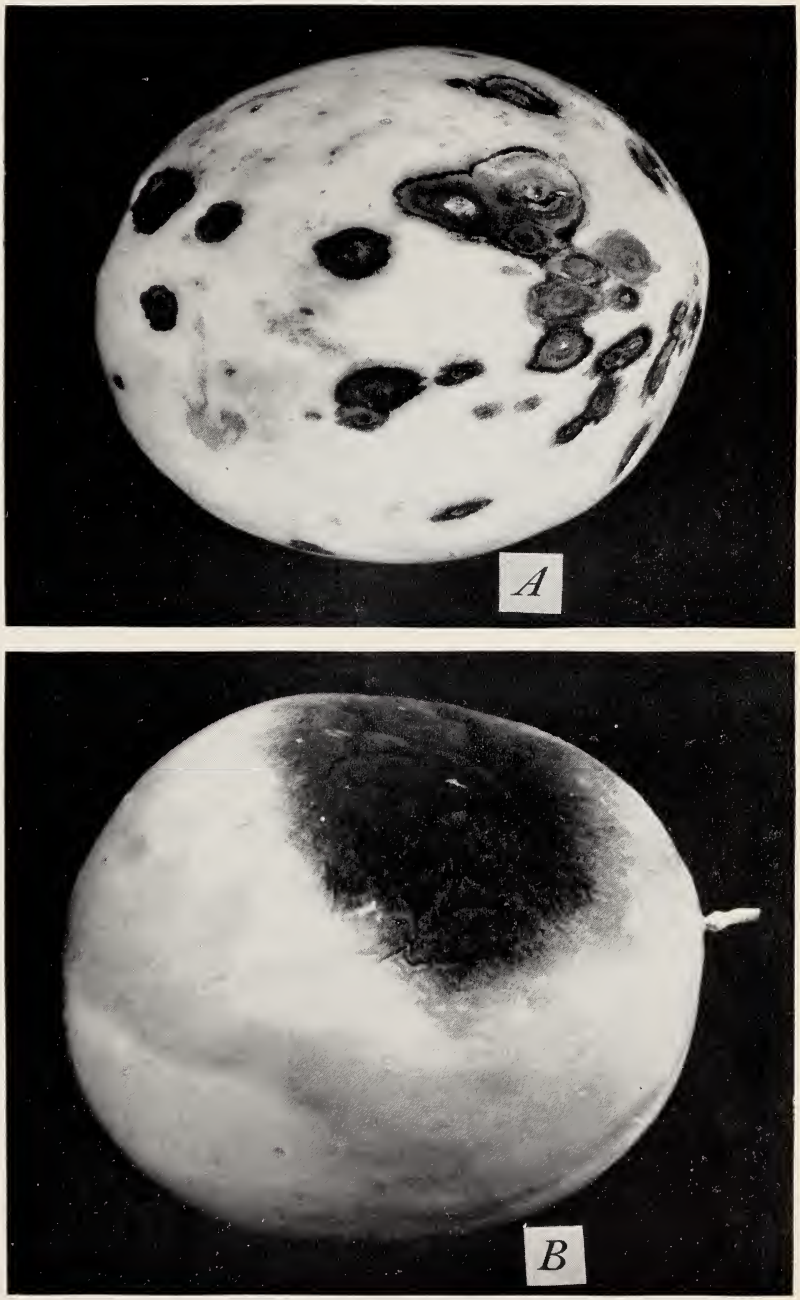
(See Cabbage, Bacterial Soft Rot, p. 5.)

BLUE MOLD ROT

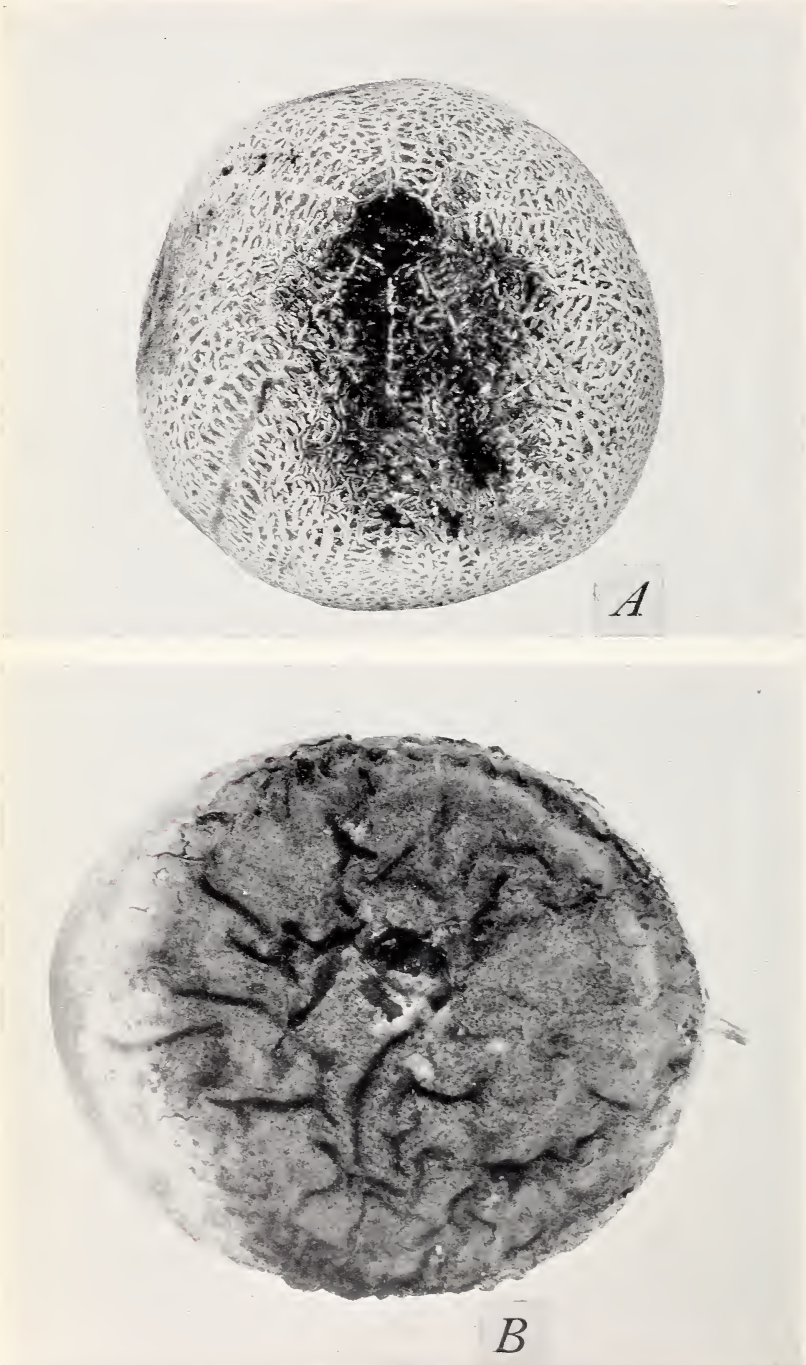
(*Penicillium* spp.)

Although blue mold rot, caused by different species of the fungus *Penicillium*, is commonly found on the market on cantaloups and Honey Dew melons it is a disease of only minor importance. It is usually found on riper melons or on those that have been held for some time at the market. It frequently occurs as a secondary invader of Honey Dew melons that are affected with low temperature breakdown (pl. 13).

The disease is recognized by the characteristic light-blue (in some cases light-green) spore masses that develop abundantly over the lesions (*206*).



A, Alternaria rot of Honey Dew melon; *B*, charcoal rot of Honey Dew melon.



A, Cladosporium rot of cantaloup; *B*, diplodia rot of Honey Dew melon.

CHARCOAL ROT

(*Rhizoctonia bataticola* (Taub.) Butler)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Charcoal rot has been found as a fruit rot of cantaloups in the field in Texas. Only in a few instances has it been observed as a market disease of domestic muskmelons. However, the disease occurs abundantly in the shipments of Honey Dew melons from South America and is responsible for considerable loss in some lots.

Charcoal rot in early stages on Honey Dew melons develops as a gray or purplish-gray water-soaked spot that rapidly enlarges at favorable temperatures so that a single lesion may eventually cover the entire surface of the melon. As the decay advances the causal organism forms a thin crust of black, tightly packed, minute sclerotia over the surface of the lesion. This black, wrinkled crust gives the decay a characteristic appearance (pl. 14, *B*). The crust is easily peeled off to expose the water-soaked, but more or less normal-colored, infected flesh beneath. The flesh of diseased melons has a pungent sour odor and taste. Infected melons are somewhat soft and spongy, and unless invaded by soft rot organisms they eventually dry out to form black wrinkled mummies.

CAUSAL FACTORS

Charcoal rot is caused by *Rhizoctonia bataticola*, a fungus that has long been known as the cause of the charcoal rot of sweetpotatoes under the name *Sclerotium bataticola* Taubenhaus.

The growth of the fungus in culture and the development of the decay of Honey Dew melon occur most rapidly at approximately 90° F. The maximum temperature lies somewhat above 105° and the minimum temperature near 50°.

CONTROL MEASURES

The development of the decay during transit can be held in check by temperatures of 50° F. or less (*191*, *206*).

CLADOSPORIUM ROT

(*Cladosporium cucumerinum* Ell. and Arth.)

The fungus *Cladosporium cucumerinum* is the cause of cucurbit scab, an important leaf and fruit disease of field and greenhouse cucumbers (see Scab, p. 45). The disease is also found in the field on pumpkin and to a limited extent on muskmelon. On the latter, however, the fungus is chiefly important as the cause of a market decay of cantaloups and Honey Ball and Honey Dew melons, and is known as cladosporium rot.

Cladosporium rot commonly develops at the stem scar of cantaloups and Honey Ball melons. It is recognized there as a shallow layer of decayed tissue that can be easily slipped off the stem scar with the fingers. It is also frequently found developing along the skin cracks of Honey Ball melons particularly where paper wraps have been used. Side lesions on cantaloups are somewhat irregular in outline with indefinite edges (pl. 15, *A*). Here also the diseased rind and tissues

23735°—38——4

immediately beneath may be slipped off the melon. On Honey Ball melons the lesions are more definite in outline, frequently with fimbriated edges.

On Honey Dew melons cladosporium rot is recognized by the numerous oval black spots that range in size from mere specks to lesions an inch or more in diameter and develop in profusion over the surface of the melons (pl. 13). They at first are shiny and black because of development of the fungus beneath the epidermis. Following the rupture of the epidermis or the placing of the melon under humid conditions the lesions soon become covered with a velvety dark-green layer of abundantly sporulating mold. The decay at times resembles alternaria rot, but is distinguished by the presence of the dark-green mold that later turns black, by the shallow nature of the decay that is confined chiefly to the rind, and by the absence of brown colors on the white-skinned varieties. On cantaloups it may be easily confused with alternaria rot, particularly in later stages when the color of the mold has changed from dark olive-green to nearly black.

The causal fungus grows best near 70° F.; the maximum temperature lies between 90° and 95°. The fungus grows slowly but continues to develop at 37°. Cladosporium decay frequently develops in stored melons at temperatures as low as 32° to 34°. The decay is favored by high humidity.

The most important measures for the control of cladosporium rot are good refrigeration in transit (45° to 50° F.) and prompt handling following the arrival of the melons on the market. The discontinuance of the practice of wrapping cantaloups and Honey Ball melons or the prompt removal of the wraps by the retailer has been recommended (*158*, *206*).

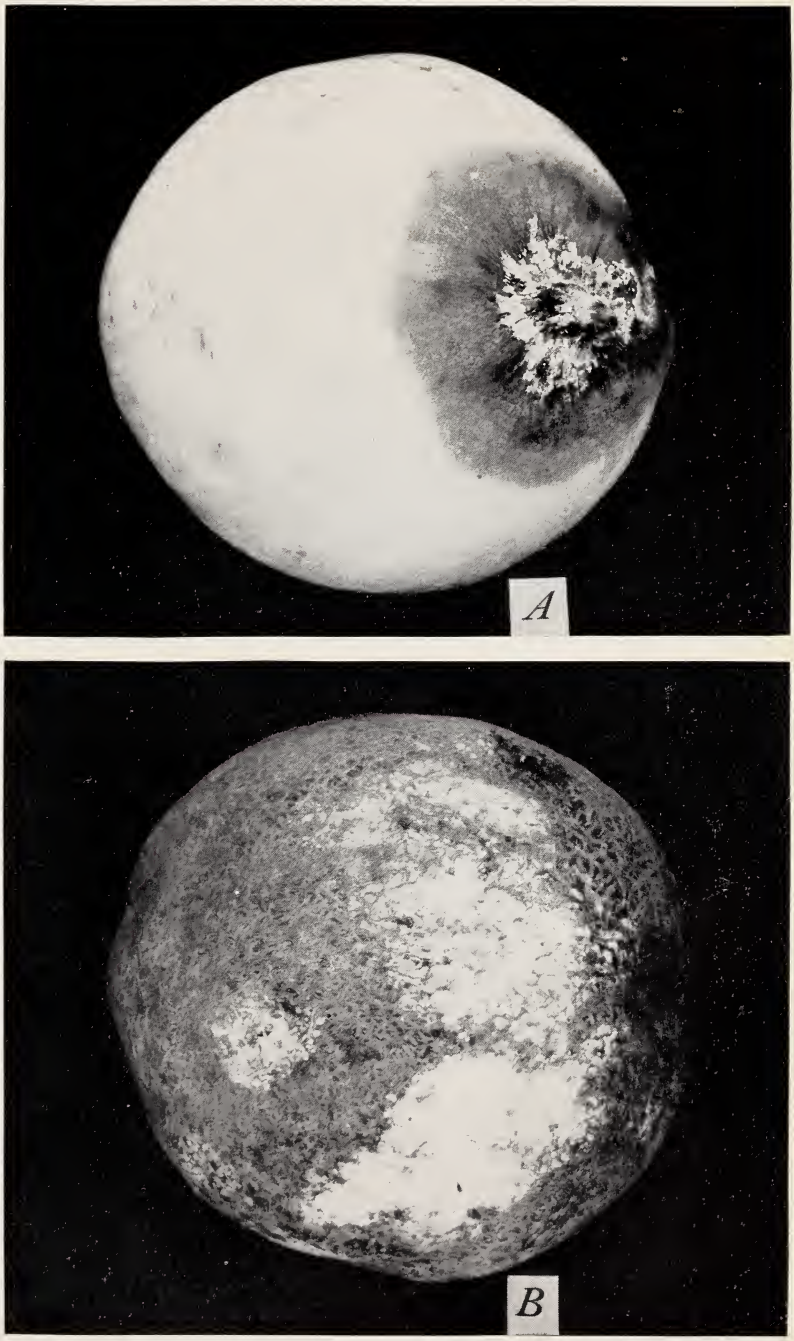
DIPLODIA ROT

(*Diplodia natalensis* Ev.)

Diplodia rot has been reported as a field decay of cantaloup and cucumber fruits in Texas. Diplodia stem end rot has long been recognized as a serious decay of watermelons (see Watermelon, Stem End Rot, p. 62).

Under market conditions diplodia rot is found only on Honey Dew melons from South America. It is characterized by a bleaching of the skin and the development of buff-colored lesions that may occur anywhere over the surface of the melons, but rarely at the stem end. Usually there is only one lesion on a melon, but by enlargement this may eventually cover most of the melon surface. In later stages the lesion becomes black in the center, the dark color finally spreading outward over most of the lesion. The decayed tissues are spongy and somewhat softened and have a pungent sour odor and taste, but the melon retains its normal shape. In later stages the decay, occurring as it does under the same conditions as those in which charcoal rot is found, may be mistaken for the latter. It is distinguishable, however, by the development of a greenish-black stroma beneath the epidermis which later turns black. The coarse black fruiting bodies of the causal organism develop in profusion over it and rupture the epidermis which peels back and curls up (pl. 15, *B*).

The causal organism and the decay produced by it develop most rapidly between 85° and 90° F. The maximum temperature lies near 100°, and the minimum between 45° and 50°.



A, Pink mold rot of Honey Dew melon; *B*, fusarium rot of cantaloup.

Fusarium rot of Casaba melon.

The disease can be held in check during transit by temperatures of 50° F. or lower. As a further measure of control all mechanical injury should be avoided during picking and packing operations (*192*, *193*, *206*).

FUSARIUM ROT

(*Fusarium semitectum* Berk. and Rav.; *F. equiseti* (Cda.) Sacc. var. *bullatum* (Sherb.) Wr.; *F. scirpi* Lamb. and Fautr.; *F. scirpi* Lamb. and Fautr. var. *acuminatum* (Ell. and Ev.) Wr.; *F. scirpi* Lamb. and Fautr. var. *compactum* Wr.; and other species.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Fusarium rot is one of the most common diseases of muskmelons on the market. It is found on cantaloups, Honey Ball, Honey Dew, Persian, Spanish, Casaba, and other melons. It is of most importance on fully ripe melons. Although on domestic melons the lesions may occur anywhere over the surface, on Honey Dew melons from South America the disease is found chiefly as a stem-end decay. It is also found affecting cucumbers on the market.

On green-skinned varieties of melons, fusarium rot frequently occurs as small scattered lesions of varying size and shape, with numerous small lesions commonly coalescing to form large diseased areas. The most conspicuous external symptom is the development over the lesion of the white or pinkish-white mycelium of the causal organism (pl. 16, *B*). Upon cutting the melon open the diseased tissues are seen to be held together by the fungus mycelium to form sharply delineated spongy bodies that can easily be lifted out intact from the healthy flesh. They may either be confined to the outer tissues of the melon immediately beneath the rind or they may extend to the seed cavity (pl. 17).

On the white-skinned varieties the lesions are brown at first and form more sharply defined spots than on the green-skinned varieties. They likewise later become covered with white or pinkish-white mold. The internal symptoms are similar to those on the green-skinned varieties. Occasionally the purplish-red discoloration produced by *Fusarium scirpi* var. *acuminatum* (and certain other species) is evident even before cutting the melon (pl. 13). The decay produced by this species is further characterized by the extremely bitter taste of the infected tissues.

CAUSAL FACTORS

Studies on the New York market have shown that the fusarium rot of muskmelons is caused chiefly by *Fusarium semitectum*, *F. equiseti* var. *bullatum*, *F. scirpi*, *F. scirpi* var. *acuminatum*, and *F. scirpi* var. *compactum*, but that *F. graminum* Cda., *F. semitectum* Berk. and Rav. var. *majus* Wr., *F. equiseti* (Cda.) Sacc., *F. culmorum* (W. G. Smith) Sacc., *F. moniliforme* Sheldon var. *subglutinans* Wr. and Rg., and *F. solani* (Mart. pr. p.) App. and Wr. are also involved.

The symptoms produced by the various species are strikingly similar with the following exceptions: *Fusarium scirpi* var. *acuminatum* produces a purplish-red discoloration of the diseased flesh, which has a very bitter taste; *F. culmorum*, *F. graminum*, and *F. moniliforme* var. *subglutinans* produce some reddish discoloration but do not produce the bitter flavor.

The temperature relations of the various species are also similar, most of them having optimum temperatures of either 75° or 80° F. (85° in one case). The upper limit for growth in 1 week lies between 95° and 100°. In most cases growth is slight at a temperature of 37° or lower.

Fusarium argillaceum (Fr.) Sacc., *F. orthoceras* App. and Wr., and *F. reticulatum* Mont. have been isolated from decaying cucumbers.

CONTROL MEASURES

The most important methods for controlling fusarium rot are the avoidance of wounds during picking and packing operations, good refrigeration in transit, and prompt handling of the melons after their arrival on the market (*106*, *123*, *157*, *158*, *206*).

LOW TEMPERATURE BREAK-DOWN

Honey Dew melons stored for 2 weeks or longer at temperatures of 32° to 34° F. (or, under certain conditions, even at higher temperatures), either before shipment or after their arrival on the eastern markets, frequently become affected with low temperature break-down. There may either be no evidence of injury to the melons at the time of their removal from the car or there may be an irregular water-soaking of the rind. After the melons have been removed from storage and held at room temperatures for a day the typical symptoms develop. The injury is first manifested by water-soaking of the rind which may be restricted to limited areas of irregular size and shape or it may extend over half or more of the melon surface (pl. 18). The juices of the melon exude and the skin becomes sticky. There is a gradual softening of the melon although the tissues retain their general form and structure. The affected rind may eventually become drab gray or somewhat brown.

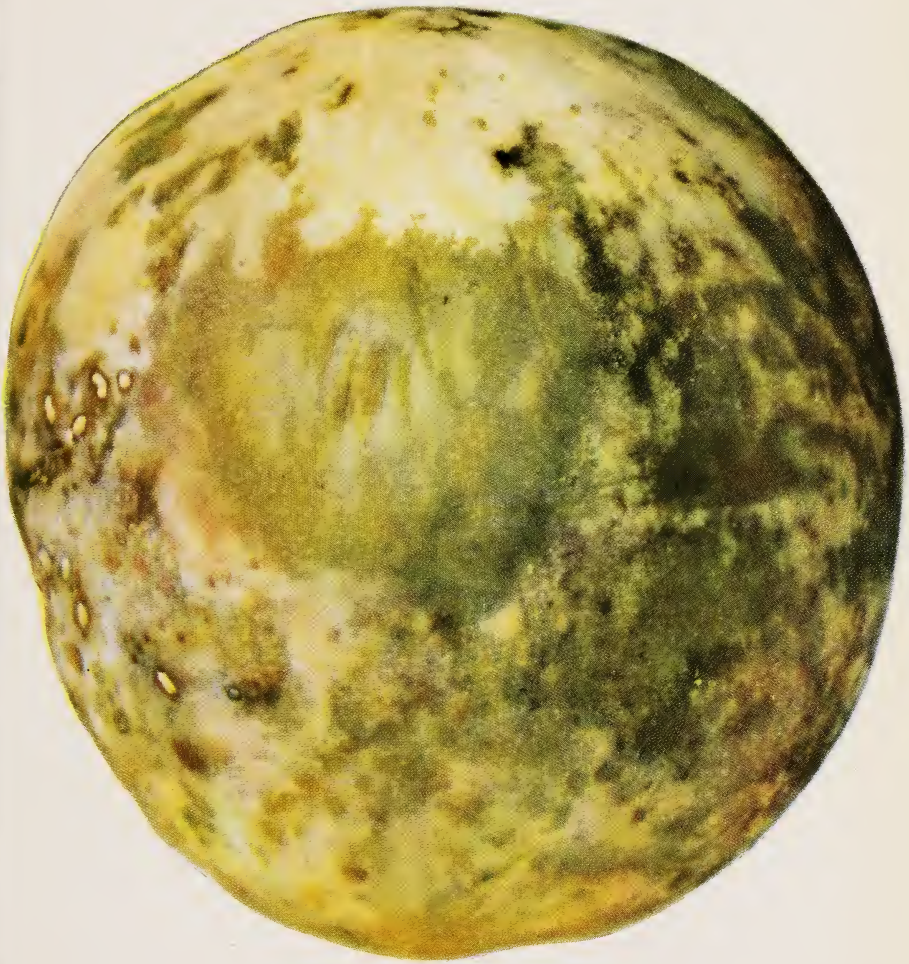
Low temperature break-down is usually quickly followed by various decays including chiefly alternaria rot, blue mold rot, cladosporium rot, and fusarium rot (pl. 13).

Experiments are under way to determine exactly under what conditions Honey Dew melons may be stored to avoid losses from low temperature break-down. Until such information is available it must be held in mind that melons stored for any considerable length of time may be subject to this type of injury.

MACROSPORIUM LEAF BLIGHT

(*Macrosporium cucumerinum* Ell. and Ev.)

Macrosporium leaf blight is one of the most important field diseases of the muskmelon. It also occurs on cucumber, squash, and watermelon. It is a market factor on cucurbit fruits only in an indirect way, except occasionally on muskmelons, where it may cause considerable spotting. It usually involves only the foliage, but frequently this is so severely spotted that the vines become defoliated and as a result the yield is decreased, and poorly sized and poorly flavored fruits develop. Sometimes the defoliation leads to sunburn of the fruits.



Low temperature break-down of Honey Dew melon.

The young lesions are at first circular, very small, and water-soaked in appearance. As they enlarge they remain circular but develop the concentric rings that are characteristic of *Macrosporium* and *Alternaria* lesions on leaves. The margins of the spots are definite on the upper side of the leaf, but indefinite on the lower side. The lesions may remain single and enlarge to one-half inch in diameter, or they may coalesce and cover as much as one-fourth of the leaf surface. The lesions appear first on the older leaves near the center of the hill. The foliage of the muskmelon suffers more severely than that of other susceptible cucurbits; often complete defoliation occurs. (See Alternaria rot, p. 46.)

This disease is caused by a fungus commonly referred to in the literature as *Macrosporium cucumerinum* (Ell. and Ev.), although the name *Alternaria brassica* (Berk.) Sacc. var. *nigrescens* Peglion is occasionally used. More recent studies indicate that the fungus should probably be known as *A. cucumerinum* (Ell. and Ev.) J. A. Elliott.

The causal agent overwinters in infested plant debris. Spores carried over winter in Georgia on infected seed and in infested soil retained their viability. This suggests the advisability of seed treatment. The usual control practices are crop rotation and spraying with bordeaux mixture or dusting with copper-lime dust (*92*, *122*, *134*, *148*, *156*, *169*, *189*, *208*).

PHYTOPHTHORA ROT

(*Phytophthora* sp.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Phytophthora rot has been reported only on Honey Dew melons. The disease is regularly responsible for appreciable losses in the shipments from South America. Ordinarily it is of minor importance on domestic muskmelons, occurring chiefly on a few individuals scattered through occasional carlots originating in Colorado and California. On several occasions, however, the disease has been responsible for the loss of practically the entire contents of the car.

The decay may occur in the form of one or several slightly sunken spots of practically normal color except for a limiting border of reddish brown. Generally, however, it is recognized by the presence of an irregular bleached area of the rind that is not sunken nor well defined in extent (pl. 19, *A*). As the lesion enlarges the affected flesh becomes slightly softer and more spongy than normal. This together with the slight bleaching are the only external evidences of the disease. The affected area of the melon later becomes somewhat water-soaked under pressure. The epidermis loosens, forms blisters, and is easily torn away. As the lesion enlarges, covering most of the surface, the fruit shrivels, the rind wrinkles and folds, and the melon flattens out somewhat under its own weight. Under humid conditions a wet appressed dirty white felty mycelial growth covers the lesion (pl. 19, *B*). Diseased flesh, although practically normal in color, is slightly sour. When affected flesh is pressed between the fingers the juice is squeezed out, leaving a felty residue of fungus mycelium which has formed in abundance.

CAUSAL FACTORS

It has not been determined whether more than one species of *Phytophthora* is responsible for this decay. Artificial inoculations with several different species have produced identical symptoms. The temperature relations of the isolates obtained from widely different sources were very similar. All grew most rapidly and produced decay most abundantly at a temperature of 80° to 85° F. The maximum temperature lay between 95° and 100° and the minimum somewhat below 50°. The fungus undoubtedly is soil borne and infects the melons while they are still attached to the vines. The exact conditions under which extensive infection occurs in the field have not been determined.

CONTROL MEASURES

Until more is known regarding the conditions favoring infection the only control measure that can be recommended is that of maintaining transit temperatures of 50° F. or lower (*119*, *206*).

PINK MOLD ROT

(*Cephalothecium roseum* Cda.)

Pink mold rot caused by the fungus *Cephalothecium roseum* is found on the market occasionally on cantaloups and Spanish melons and more frequently on Honey Dew melons. It is an important decay only in shipments of Honey Dew melons from South America. The decay is also occasionally seen on cucumbers.

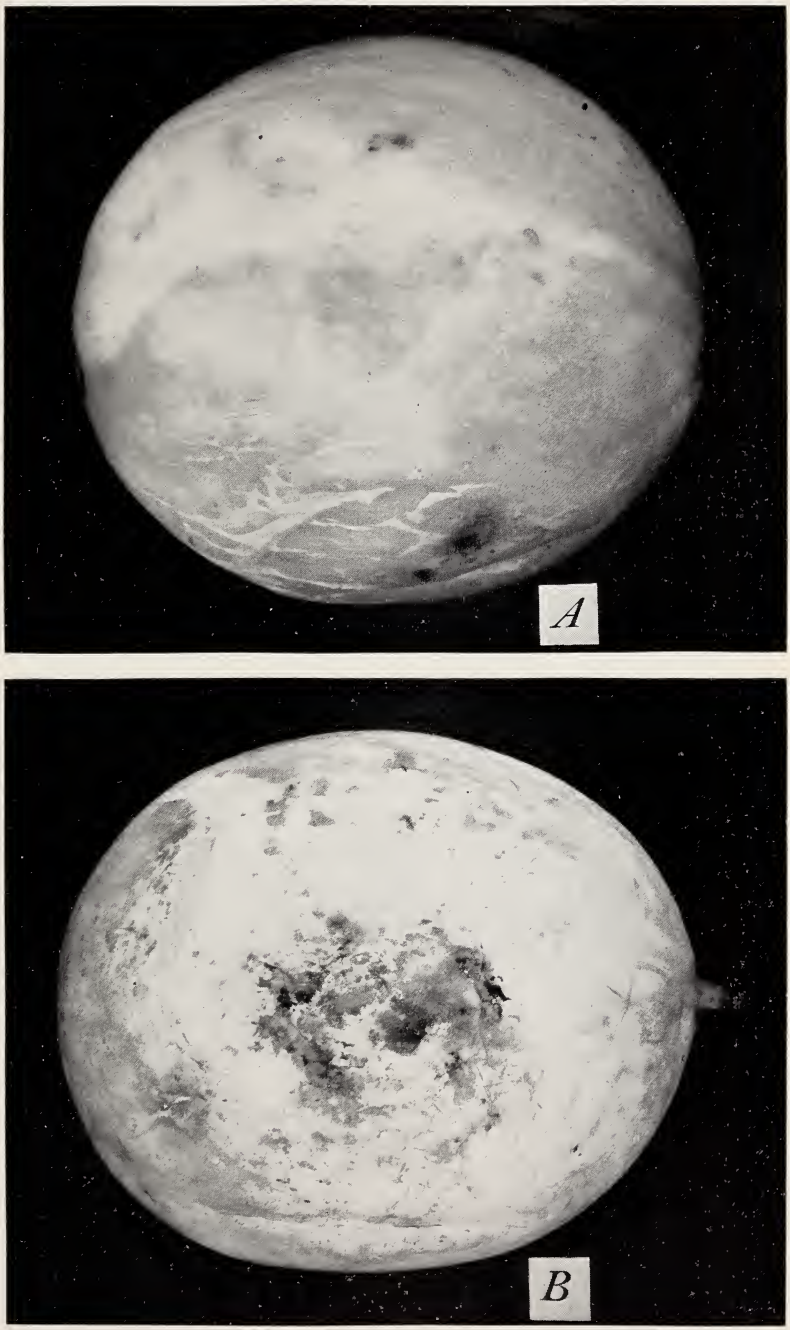
The earliest symptom of pink mold rot on Honey Dew melons is a brown discoloration of the rind. Lesions vary from small spots 1 inch in diameter to large areas covering one-third of the melon surface. Frequently these occur at the stem end (pl. 16, *A*); later the lesion becomes covered with the mycelium and the abundant pink spore masses of the causal fungus. The decay at times resembles some of the fusarium rots but can easily be distinguished from them by the extremely bitter taste of the affected flesh.

The causal organism grows most rapidly at a temperature of about 70° F. The highest temperature at which growth is made lies somewhat above 95°; the lowest somewhat above 40°. Control measures therefore include refrigeration in transit to near the latter temperature (*106*, *123*, *206*).

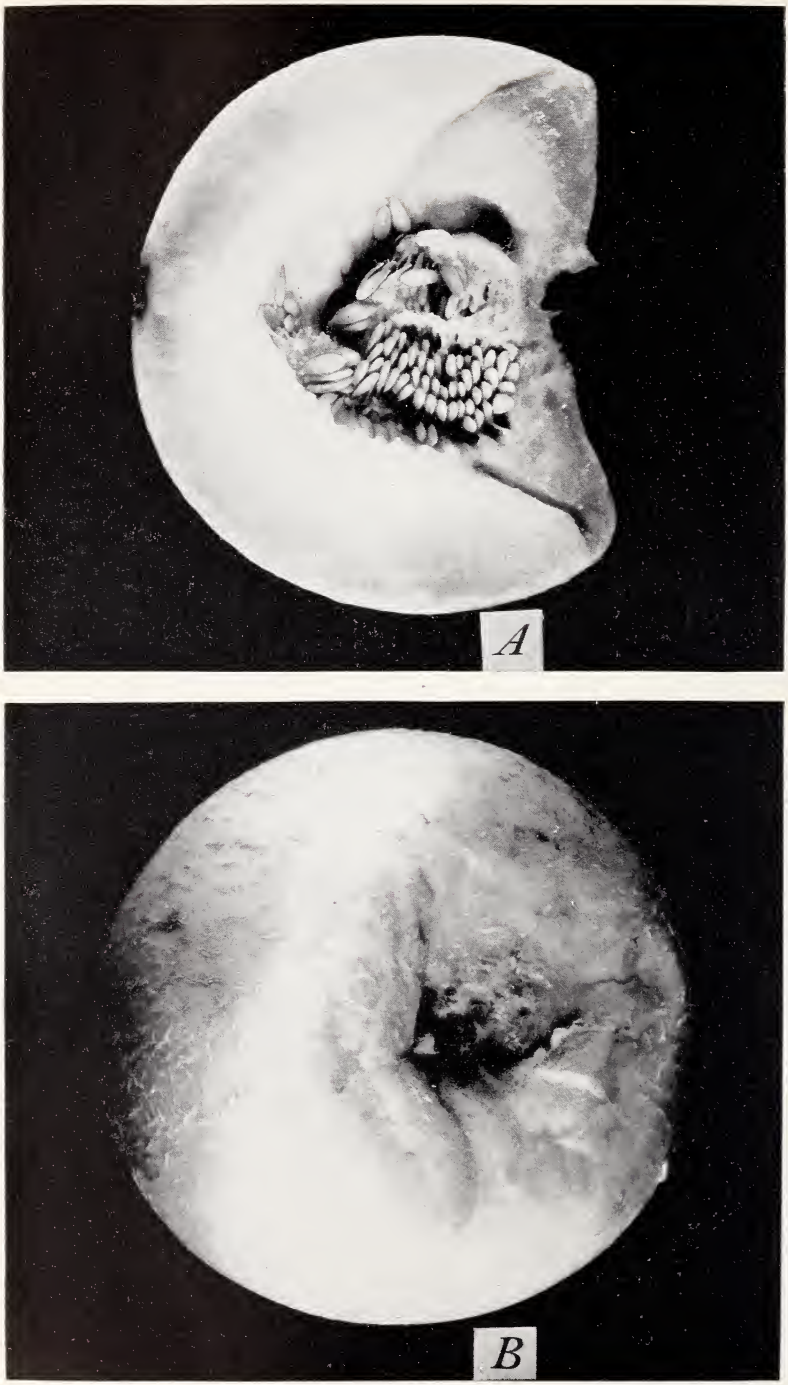
POWDERY MILDEW

(*Erysiphe cichoracearum* DC.)

Powdery mildew has long been known as a disease of minor importance on cucumber, muskmelon, pumpkin, squash, watermelon, and other cucurbits. Occasionally it is a serious problem in the greenhouse cucumber crop. In 1925, however, the disease appeared in epiphytotic form on the cantaloup crop of the Imperial Valley, Calif., causing an estimated loss of 15 percent of the crop. The following year it was even more destructive, causing a loss of from 25 to 35 percent of the crop, and it has varied in severity since then. It has likewise become of increasing importance in Arizona and Georgia.



A, Phytophthora rot of Honey Dew melon, early stage; *B*, phytophthora rot of Honey Dew melon held under humid conditions.



A, Internal view of a Honey Dew melon showing rhizopus soft rot; *B*, external view of a Honey Dew melon showing rhizopus soft rot.

Rhizopus rot of squash.

The disease is recognized by the presence of the causal fungus, *Erysiphe cichoracearum*, one of the powdery mildews, upon the leaves. Tiny patches of white mold first appear on the stems and leaves, particularly at the crown of the plant. Although they are more numerous on the shaded portions of the stems and the under sides of the basal leaves, they soon appear on the stem and on both surfaces of leaves on all parts of the plant. The spots enlarge and coalesce, covering the affected areas with a powdery growth. Affected leaves turn pale yellowish green and yellow, later wilting, drying, and falling off. Severely diseased plants die prematurely, exposing the melons to sunburn and eventual decay; they also produce melons of inferior quality and flavor. In this indirect way the disease has been of great importance on the market, brought about in part by the practice of harvesting the crop as early as possible after the mildew appears in order to avoid loss, with the result that many melons of distinctly low quality have been placed on the market.

In the Imperial Valley the organism overwinters on volunteer melon vines and on other cucurbits, chiefly the squash, that are grown throughout the winter months.

Powdery mildew can be controlled on greenhouse cucumbers by various dusts and sprays. However, in the field applications of fungicides have not been very successful on muskmelons. Marked progress has been made in the development of resistant varieties of cantaloups suitable for use in southern California, where in 1936 Powdery Mildew Resistant Cantaloup No. 45 was planted on nearly half the acreage in Imperial Valley, with a considerable increase in 1937 (*136*, *145*, *146*, *147*, *167*).

RHIZOPUS SOFT ROT

(*Rhizopus nigricans* Ehr., and *Rhizopus* sp.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Rhizopus soft rot is of common occurrence on all types of muskmelons found on the market. It is a rapidly developing and destructive decay. The earliest symptoms are a softening and a pronounced water-soaking of the flesh. As the lesions enlarge the affected flesh is readily flattened by the weight of the melon or pushed in by adjacent melons in the container (pl. 20, *B*). Nevertheless, it holds together, and the affected flesh is never completely disintegrated and soupy as is that of melons affected with bacterial soft rot. The skin may be torn or remain unbroken for some time. There is often no external evidence of the causal organism, although frequently a slight development of mycelium may occur here and there on the lesion. Internally the decay is recognized by the soft water-soaked flesh that is sharply delimited from the healthy tissue and can usually be removed intact (pl. 20, *A*). It has a slightly sour odor and taste. The coarse mycelium of the causal organism can be demonstrated by gently pulling apart the diseased tissue.

The decay is also found on cucumbers, squashes (pl. 21), and watermelons. In a general way the symptoms are similar to those described above for the decay of muskmelons.

CAUSAL FACTORS

Rhizopus nigricans has generally been assumed to be the cause of the decay of cucurbits and other vegetables. However, it has been shown by artificial inoculation that a number of different species of *Rhizopus* are capable of producing a decay of cantaloups, cucumbers, squashes, and watermelons. Studies of a large number of isolates from muskmelons indicate that about half of them belong to the low-temperature group typified by *R. nigricans* and the other half to the medium-temperature group. *R. arrhizus* has been reported from pumpkin.

The spores of *Rhizopus* are widely distributed in the soil, water, and air; consequently infection may occur at any time. Wounds of some sort appear to be essential for the entrance of the organism. The loss is usually greater on the riper melons. The temperature requirements differ considerably with the different species. For *Rhizopus nigricans* and others of the low-temperature group, growth on artificial media is greatest at 70° to 85° F. Those isolated from decaying Honey Dew melons grew most rapidly in culture and caused decay in melons most rapidly at 80° F. Those species of the intermediate-temperature group are most active at 85° to 95° F.

CONTROL MEASURES

The chief methods of controlling rhizopus soft rot are the avoidance of bruising and injuring melons and other cucurbits during the picking and packing operations, the use of refrigeration (40° to 45° F.) in transit, and prompt handling of the products upon their arrival at the terminal markets (*106*, *137*, *158*, *205*, *206*).

PUMPKINS

The pumpkin of commerce is the large, globular, or oblong fruit of the pumpkin plant (*Cucurbita pepo* L.). The marketability of pumpkins depends upon their color, maturity, and freedom from decay.

The important field diseases affecting the vines, and indirectly the fruits, are downy mildew (p. 42), mosaic (p. 43), powdery mildew (p. 54), and bacterial wilt (p. 40).

The following diseases occur both on the vines and the fruit in the field: Mycosphaerella black rot or gummy stem blight, scab, and sclerotinia stem and fruit rot.

Fusarium rot (p 51) and rhizopus soft rot (p. 55) are important transit and market diseases.

SQUASH

The term squash is applied loosely to the fruit of various members of the gourd family, but is not limited to any one species. The summer squashes are fruits of varieties of *Cucurbita pepo* (p. 36), and the winter squashes are fruits of other species (*C. maxima* Duch.). The summer squashes are eaten while the rind is still soft; consequently, more careful handling is required for these fruits than for the varieties not harvested until the rinds have become mature and hard. The marketability of all varieties depends upon freedom from bruising and disease.

There are several diseases that affect fruit production and also the quality of the fruits by reduction of the vitality or killing of the vines. Bacterial wilt (p. 40) and fusarium wilt (p. 60) affect the vascular system and roots; while anthracnose (p. 37), bacterial leaf spot (*Bacterium cucurbitae* Bryan), blue mold rot (p. 48), cladosporium rot (p. 49), fusarium rot (p. 51), mycosphaerella rot (p. 60), downy mildew (p. 42), mosaic (p. 43), powdery mildew (p. 54), and septoria leaf spot occur on the foliage. Anthracnose, choanephora fruit rot (*Choanephora cucurbitarum* Berk. and Rav.) Thax., gray mold rot (*Botrytis* sp.), and rhizopus soft rot (p. 55) are important diseases of the fruits (*94*).

CHOANEPHORA FRUIT ROT (BLOSSOM ROT)

(*Choanephora cucurbitarum* (Berk. and Rav.) Thax.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

This disease seems to be of importance only upon squash, although it has been observed on the flowers of cucumber, althea, scarlet hibiscus, okra, cotton, and a few other plants. Blighting of the blossoms and a subsequent decay of the fruits take considerable toll in the market garden districts. Although a fruit rot is produced in the field, apparently few, if any, affected fruits are shipped to market.

The attack on the flowers is usually evident the second day after their opening. The fading blossoms are covered with a dense growth of immature, white, sport-bearing structures of the fungus that change to brown and finally purplish black at maturity. From the flower the mycelium works down into the newly forming fruits and causes decay. When larger fruits become affected at the blossom end, a rapidly progressing, soft, wet rot is produced. Half of a moderate sized fruit may rot within 15 to 20 hours. A profusion of black sporangia develops on the decaying fruit, giving it a very characteristic appearance.

CAUSAL FACTORS

The causal fungus, *Choanephora cucurbitarum*, looks like *Rhizopus* when fruiting, but the absence of coarse, whiskerlike mycelium serves to distinguish it from that fungus. The spores are disseminated by insects, rain, and wind. Thick-walled spores (zygospores) on the surface or within decaying plant tissue enable the fungus to withstand drought and extremes of temperature. This fungus causes greatest damage in seasons of high humidity and excessive rainfall.

CONTROL MEASURES

Since spores of this fungus are scattered largely by insects, spraying for the striped and spotted cucumber beetles affords one means of control (*207*).

MYCOSPHAERELLA BLACK ROT (MYCOSPHAERELLA WILT; GUMMY STEM BLIGHT)

(*Mycosphaerella citrullina* (C. O. Smith) Gross.)

The fungus (*Mycosphaerella citrullina*) which attacks a number of different cucurbits (p. 36) causes a dry rot of the squash. The rind

usually remains normal, but eventually the entire flesh becomes a mass of black diseased tissue (*150*).

WATERMELON

The watermelon (*Citrullus vulgaris* Schrad.) is a member of the gourd family (Cucurbitaceae). Its marketability depends upon size, shape, color, and freedom from blemishes and decay.

The watermelon is subject to a number of field diseases that are not characterized by lesions on the fruit but are important from the production point of view, because they reduce the yield, and also indirectly from the market point of view, because they lead to the development of undersized, poorly shaped, flavored, and colored fruit. The most important of these are fusarium wilt (*Fusarium bulbigenum* (Cke. and Massee) var. *niveum* (E. F. Smith) Wr.) and root knot. Other diseases of this type which are of much less importance either because of their slight effect upon the plant or their limited occurrence are the leaf spots (caused, respectively, by *Cercospora citrullina* Cke., *Macrosporium cucumerinum* Ell. and Ev., *Phyllosticta citrullina* Chester, and *Septoria citrulli* Ell. and Ev.); powdery mildew; Texas root rot (*Phymatotrichum omnivorum* (Shear) Duggar); and thielaviopsis root rot.

The watermelon is practically immune to downy mildew and bacterial wilt and has only rarely been reported subject to the virus disease, mosaic, although all three of these diseases are of great importance on other cucurbits.

Some diseases occur both on the vines and the fruits. These are anthracnose (*Colletotrichum lagenarium* (Pass.) Ell. and Hals.),[9] mycosphaerella black rot, sclerotium rot, and soil rot (*Corticium vagum* Berk. and Curt.).

The following diseases occur primarily on the fruits either in the field or after they are harvested and consequently are diseases of importance in transit and on the terminal markets: Bacterial soft rot (p. 48), blossom end rots, pythium and phytophthora rots (caused, respectively, by *Pythium* spp. and *Phytophthora* spp.), rhizopus soft rot (p. 55), and stem end rot (*Diplodia* sp.).

Melons are subject to chemical injury when loaded into cars that have been used for shipment of chemicals, such as salt, fertilizers, tar products, and the like, unless precautions are taken to cover the floor and walls with protective materials. An injury similar in appearance develops when tender-skinned varieties are subjected to bruises on unprotected floors or when too much weight is placed on them due to heavy loading.

Sunscald is a factor in the field. This often results from premature death of leaves, which normally shade the fruit.

ANTHRACNOSE

(*Colletotrichum lagenarium* (Pass.) Ell. and Hals.)

OCCURRENCE, SYMPTOMS, AND EFFECTS

Anthracnose is the most serious of all field diseases of the watermelon and is more important on that crop than on either cucumber

[9] Syn. *Glomerella lagenarium* (Pass.) Stevens.

Anthracnose fruit spot of watermelon.

or muskmelon. This is in part due to the fact that the fruits must remain in the field a long time and that individual fruits have such great value. The disease occurs not only on the vines and the immature fruits, causing reduced yield, but also on the mature fruits, disfiguring them and even causing rot.

The lesions on the leaves are black rather than brown, as on most other cucurbits, and the foliage looks scorched. Petiole and stem lesions are similar to those on muskmelon in appearance and severity, causing defoliation, especially at the centers of the hills, and death of the distal portions of the vines. Premature death of the leaves exposes the fruits to the sun and to sunburn and also causes abnormal ripening of the fruit, with resultant inferior flavor.

On the young fruit the lesions are small, black, depressed spots which soon develop acervuli, the fruiting bodies of the fungus. On older fruit, the disease is recognized by the presence of elevated pimples with yellow translucent centers. On mature fruit the nailhead type of lesion is very common (pl. 22). These are small, circular, raised welts on the rind, which are dark green in color. As they increase in size the centers turn brown, become sunken, and under moist conditions the pink acervuli of the fungus are formed in which sticky masses of pink spores are produced. As the lesion enlarges the center becomes more sunken, often turns black, and eventually the surface cracks, exposing either the rotted tissue or a cavity underneath. The fungus causes a rather slowly advancing dry rot of the rind tissue and finally penetrates the pulp. Frequently a halo of lesions develops around that part of the surface that is in contact with the soil. These lesions often become black and corky. It is this kind of infection which leads to rotting in the field. Under extremely dry, hot conditions, such as occur in California during the melon harvest, the lesions may remain in the blister or nailhead stage.

The disease develops and spreads in transit, since the fungus is able to lie dormant in the melon rind and to develop visible lesions when moisture and temperature conditions become favorable.

(See Cucumber, Anthracnose, p. 37, for causal factors, control measures, and literature citations.) (*91*, *163*, *171*, *201*.)

CHEMICAL AND MECHANICAL INJURY

Watermelons frequently show friction bruises, slight water soaking, and discoloration of the rind due to mechanical injuries while in transit. These injuries are most prominent on melons in contact with the floor or walls of the car. Dark greenish-brown spots also occur on the ends of melons where they come in contact with each other or with the end of the car when the loads are badly shifted. Some of these injuries are often confused with chemical injury, but in general the affected tissues are not so water-soaked or soft and flabby as those affected by chemicals.

Chemical injury to watermelons results when they come in contact with the floor and side walls of cars used to haul certain chemical substances. Cars of melons without sufficient bedding or those in which the bedding becomes wet are especially likely to show damage if the car has an excessive amount of chemical residue on the side walls and floor. Among the chemicals shipped in freight cars it has been found that common salt and certain fertilizers, such as superphosphate, muriate of potash, nitrate of soda, sulphate of ammonia,

and sulphate of potash, are most likely to cause injury when in contact with melons. Under pressure of the load the side of the melon in contact with such chemicals becomes water-soaked, soft, flattened, and discolored brown. Lime, the chemical most commonly found in cars, is not injurious to melons.

Mechanical and chemical injuries of melons can be practically eliminated if they are loaded into a thoroughly clean car whose side walls have been lined with heavy paper and if an ample supply of dry straw bedding is provided (*131*, *132*, *133*, *171*, *201*).

FUSARIUM WILT

(*Fusarium bulbigenum* Cke. and Massee var. *niveum* (E. F. Smith) Wr.)

Fusarium wilt caused by the fungus *Fusarium bulbigenum* var. *niveum* is a serious field disease of watermelon. It is at times destructive to squash. Recently the disease has become important on muskmelons in Minnesota and New York and has been found in other States. It has been suggested that several other species of *Fusarium* are also responsible for a wilt disease of watermelon and that the squash organism is different from the one generally occurring on watermelon. Likewise the *Fusarium* from muskmelon differs in behavior from the watermelon fungus.

The disease is only indirectly a market factor. There are no lesions on the fruit, but fruit from affected vines is of inferior quality because of premature death of the vines and the burning that results from exposure to the sun.

The most prominent symptom is a sudden wilting of the plant without recovery. Generally one branch after another wilts, beginning at the tip, and dries up until all of the plant is involved. Wilting may occur at any time but is most common when the fruit is setting, the rate being largely dependent upon the air temperature, relative humidity, soil water supply, sunlight, and wind velocity. Invasion of the water vessels by the fungus is accompanied by the plugging of the vessels and by browning of these tissues, due to the toxic effect of the fungus and ends in the death of the vine.

The causal agent lives in the soil and enters the small roots from which it spreads in the water vessels. After the vines have died, the fungus may grow out to the surface and produce its spores. The fungus is disseminated by drainage water, in stable and lot manure, on tools, on the feet of men and animals, and by the wind.

Watermelons should not be grown where wilt has appeared on a previous crop. Care should be taken to prevent the drainage of water from infested to disease-free fields. Since the fungus is able to live in stable manure and other refuse, only commercial fertilizers should be used in soil infested with the disease.

Wilt-resistant varieties of watermelon have been successfully developed in Iowa, Florida, Illinois, and other States, but since there appear to be several physiological strains of *Fusarium bulbigenum* var. *niveum*, a melon variety resistant in one section may not be so elsewhere (*105*, *124*, *153*, *154*, *155*, *173*, *174*, *175*, *184*, *185*, *190*).

MYCOSPHAERELLA BLACK ROT (MYCOSPHAERELLA WILT; GUMMY STEM BLIGHT)

(*Mycosphaerella citrullina* (C. O. Smith) Gross.)

The fungus *Mycosphaerella citrullina* attacks cucumber, muskmelon, pumpkin, squash, and watermelon. Although in all cases

above-ground parts of the plant may be affected, the more important symptoms vary somewhat with the different hosts. Thus on muskmelons the stem is chiefly attacked, resulting in a wilting of the plant so that here the disease is known as "Mycosphaerella wilt." On cucumber, pumpkin, and squash the black rot of the fruit is the most important symptom (p. 44); on watermelon the disease affects chiefly the vine and is known as gummy stem blight, a name that is coming more and more into use to designate the disease on all the hosts. The disease is chiefly a field problem and has been considered only of minor importance; however, it appears that within recent years it has been increasing in importance. At times it occurs as a market disease of cucumber, squash, and watermelon.

On watermelon the main root is frequently affected just below the soil line. The disease progresses upward and outward from this point, affecting the main branches of the vine and at times the leaves. The cankers girdle the stems, thus bringing about wilting and partial killing of the vines. They are at first water-soaked, then brown, and eventually if no gum is present, ash gray in color. Frequently a reddish gummy exudate forms over the surface of the cankers and gives the characteristic symptoms suggesting the name "gummy stem blight." The black fruiting bodies (pycnidia and perithecia) of the fungus form abundantly over the surface of the cankers. Plants that are only partly killed produce poorly developed fruits of low quality.

The fungus at times causes both a blossom end rot and a stem end rot of watermelons both in the field and in transit. The rot is at first water-soaked, later turning black. The decayed tissues remain firm, and large numbers of pycnidia and perithecia develop on the rind of affected melons.

Little is known concerning the control of this disease. The two practices of treating the seed and spraying the vines appear to be at least partially effective. Crop rotation is also of value, since the fungus may live over in the soil (*135*, *138*, *150*, *164*, *171*, *201*).

PYTHIUM ROT

(*Pythium acanthicum* Drechs., *Pythium aphanidermatum* (H. A. Edson) Fitz., *Pythium artotrogus* DBy., *Pythium myriotylum* Drechs., and *Pythium periplocum* Drechs.)

AND PHYTOPHTHORA ROT

(*Phytophthora cactorum* (Leb. and Cohn) Schroet., and *Phytophthora* sp.)

Pythium rot and phytophthora rot are discussed together, inasmuch as both types of decay are similar in general symptoms and their causal organisms are very closely related.

Pythium rot is of limited economic importance. It has been observed only in excessively moist situations in the southern and midwestern watermelon growing sections. The disease is of minor importance on the market. Although the symptoms vary somewhat with the different species of *Pythium* involved, in general it may be said that they appear first as water-soaked spots that soon turn light brown, bluish brown, or chocolate brown in color. The decay progresses rapidly and the flesh becomes flaccid. In some cases when the epidermis is rubbed off the decayed tissues have a marshy odor. Under humid conditions some white surface mycelium appears.

The decay frequently appears as a blossom end rot, caused chiefly by *Pythium acanthicum*, although *P. aphanidermatum*, *P. artotrogus*, *P. myriotylum*, and *P. periplocum* are also involved.

Phytophthora rot has been reported from the irrigated watermelon-producing district of Arizona. The causal organism was determined to be *Phytophthora cactorum*. A similar if not identical decay has been observed on Winter Queen watermelons from Colorado. On several occasions during the summer of 1935, when shipments were examined on arrival at the New York market, practically the entire carlot was found decayed. In both the Arizona watermelons and those from Colorado the symptoms were similar to those of the pythium rot described above. Initially the lesions were water-soaked followed by the development of chocolate-brown colors. Usually concentric rings appeared over the affected rind; the affected flesh was spongy and had a somewhat sour odor and taste.

Little is known regarding the control of pythium rot and phytophthora rot. Undoubtedly melons that arrive at the market showing these diseases were infected in the field (*93*, *117*, *120*).

(See Cucumber, Cottony Leak, p. 41, and Muskmelon, Phytophthora Rot, p. 53.)

SCLEROTIUM ROT

(*Sclerotium rolfsii* Sacc.)

In Florida, Georgia, and South Carolina considerable loss has been suffered because of a wilting of the vines and a field fruit rot produced by the fungus *Sclerotium rolfsii*.

The wilting of the vine at first may appear somewhat similar to that caused by *Fusarium*, but closer observation shows the presence of a fine, white mycelial covering with the small, brown, spherical sclerotia of the causal organism over the base of the vine and the adjacent soil. If the soil is infested some melons are almost sure to develop decay. In practically all fruit infections, the fungus enters on the under side, usually through wounds. A small, watery discoloration surrounding the wound is the first evidence of disease. Discoloration and decay progress rapidly; in severe cases the rind cracks open and the fine, silky, white mycelium grows out in flat, fanlike radiations over the surface of the melon. Small, white, spherical sclerotia soon form in this mycelium and within a few days they appear as brown bodies resembling mustard seeds. Infected melons should not be shipped, for they will develop appreciable decay during transit.

Diseased vines should be burned and care taken to destroy as many of the sclerotia as possible at the same time. Sclerotia that are shattered off onto the soil carry the fungus over year after year. Crop rotation may help some, but is not always feasible, inasmuch as the fungus also attacks most other truck crops (*171*, *201*).

STEM END ROT

(*Diplodia* sp.)

Stem end rot probably has caused greater loss in transit than any other decay that affects watermelons. Although it also occurs on melon fruits in the field, it is of minor importance there. The pathogen does not attack fruit except through wounds or through lesions produced by other organisms.

Stem end rot of watermelon.

The first symptoms of the disease are a browning and shriveling of the stem, so that it feels hollow when pressed. Decay sets in at the point of attachment of the stem to the fruit and consists of softening and water-soaking of the tissues. As it progresses into the melon the affected tissues become brown and shriveled. In advanced decay the shriveling becomes pronounced and there is an abundant development of black pycnidia and dark-gray mycelium (pl. 23). Whitish spore masses may often be seen discharging from the pycnidia. The lesions usually start at the stem end, but they may also occur at wounds and, in the field, at the blossom end.

CAUSAL FACTORS

The causal agent, *Diplodia* sp., is a fungus, identical with or a close relative of a species of *Diplodia*, that attacks many other plants, including cucumber, squash, and muskmelon fruits (see Muskmelon, Diplodia Rot, p. 50). It is abundant on ripe or decaying vegetation in or near watermelon fields. From these sources the spores of the fungus reach the cut ends of the stems of cull melons or the blossom ends of imperfect melons. In a short time it produces spores which serve to infect sound melons through the wound made in cutting the stem, through other wounds or bruises, through anthracnose lesions, rabbit injury, and punctures made by the pickle worm. Infection can also take place in transit from spores lodged in the bedding or produced on infected melons. The cut stem is the ideal place of entry for the causal organism, because the sap which exudes serves to catch the wind-borne spores and provides a good food supply for the fungus.

The decay progresses rapidly. At temperatures between 75° and 90° F. the fungus will grow through a stem three-fourths of an inch long and decay the fruit to a line about 1½ inches distant from the base of the stem in 90 hours. It is not surprising, therefore, that melons appearing sound at the time of loading may show severe rot after being in transit 4 to 5 days.

CONTROL

The surest way to control stem end rot is to prevent germination of the spores of the causal agent in the cut stems. This involves reduction of the number of spores in the field and car and treatment of the stem end with a disinfectant.

Since the causal agent lives on all sorts of plant debris, it is important that all weeds be cut and burned. Deep plowing to bury all vegetation is helpful. During the melon-growing season all fields should be inspected once a week and all imperfect or cull melons should be carried out and fed to hogs or otherwise disposed of. The carts used for hauling the melons should be washed with a 2-percent solution of bluestone. The spray used for anthracnose is helpful, because it cuts down the number of dead leaves that provide a growing place for the fungus.

In harvesting, all decayed melons should be avoided. If a diseased one is cut into, the knife should be sterilized in a 2-percent formaldehyde solution. The melons should be cut with the longest possible stems.

When the melons are placed in the car, the stems should be recut and disinfecting paste should be applied to the cut surface. The

disinfectant should contain 6 percent of bluestone as a fungicide. A gallon of this paste can be prepared as follows: 3½ quarts of water and 8 ounces of bluestone should be brought to boiling in an enamelware kettle (iron or tin becomes corroded). While this solution is heating, 8 ounces of laundry starch should be mixed with 1 pint of cold water and stirred until a milky solution free from lumps is obtained. When the bluestone is dissolved and the solution is boiling hot the starch mixture should be added, stirring it, so as to avoid lumps. Boiling and stirring should be continued until the mixture thickens evenly (*162*, *171*, *192*, *193*, *194*, *196*, *201*).

LITERATURE CITED

CRUCIFERS

(1) Atkinson, G. F.
1893. œdema of the tomato. N. Y. (Cornell) Agr. Expt. Sta. Bull. 53, pp. [101]–128, illus.

(2) Bergey, D. H., and others.
1930. bergey's manual of determinative bacteriology; a key for the identification of organisms of the class schizomycetes. Ed. 3, 589 pp. Baltimore.

(3) Blank, L. M.
1932. the pathogenicity of fusarium conglutinans wr. at low soil temperatures. Phytopathology 22: 191–195.

(4) Bonde, R.
1930. the cabbage maggot as a disseminating agent of bacterial rots in the cruciferae. (Abstract) Phytopathology 20: 128.

(5) Brown, N. A., and Harvey, R. B.
1920. heart rot, rib rot, and leaf spot of chinese cabbages. Phytopathology 10: [81]–90, illus.

(6) Chupp, C.
1917. studies on clubroot of cruciferous plants. N. Y. (Cornell) Agr. Expt. Sta. Bull. 387, pp. 419–452, illus.

(7) ———
1928. club root in relation to soil alkalinity. Phytopathology 18: 301–306, illus.

(8) ———
1930. the effects of potash and phosphorus on tip burn and mildew of cabbage. Phytopathology 20: 307–318, illus.

(9) ———
1935. macrosporium and colletotrichum rots of turnip roots. Phytopathology 25: 269–274, illus.

(10) Clayton, E. E.
1929. studies of the black-rot or blight disease of cauliflower. N. Y. State Agr. Expt. Sta. Bull. 576, 44 pp., illus.

(11) ———
1930. a study of the mosaic disease of crucifers. Jour. Agr. Research 40: 263–270, illus.

(12) Clinton, G. P.
1907. notes on fungous diseases, etc., for 1906. turnip, brassica rapa. Conn. Agr. Expt. Sta. Ann. Rept. (1906) 15: 330.

(13) Cotton, A. D.
1922. fungus diseases of crops, 1920–1921. [Gt. Brit.] Min. Agr. and Fisheries Misc. Pub. 38, 104 pp.

(14) Cunningham, G. C.
1912. the comparative susceptibility of cruciferous plants to plasmodiophora brassicae. Phytopathology 2: 138–142.

(15) Cunningham, G. H.
1927. dry rot of swedes and turnips: its cause and control. New Zeal. Dept. Agr. Bull. 133, 51 pp., illus.

(16) Dana, B. F., and McWhorter, F. P.
1932. mosaic disease of horse-radish. (Abstract) Phytopathology 22: 1000–1001.

(17) DAVIS, W. H.
1925. DROP OF CHINESE CABBAGE AND COMMON CABBAGE CAUSED BY SCLEROTINIA SCLEROTIORUM (LIB.) MASSEE (SCLEROTINIA LIBERTIANA FCKL.). Phytopathology 15: [249]–259, illus.

(17a) DEARBORN, C. H., THOMPSON, H. C., and RALEIGH, G. T.
1937. CAULIFLOWER BROWNING RESULTING FROM A DEFICIENCY OF BORON. Proc. Amer. Soc. Hort. Sci. 34: 483–487.

(18) DRECHSLER, C.
1919. COTYLEDON INFECTION OF CABBAGE SEEDLINGS BY PSEUDOMONAS CAMPESTRIS. Phytopathology 9: [275]–282, illus.

(19) ———
1925. PYTHIUM INFECTION OF CABBAGE HEADS. Phytopathology 15: [482]–485, illus.

(20) EDSON, H. A.
1915. RHEOSPORANGIUM APHANIDERMATUM, A NEW GENUS AND SPECIES OF FUNGUS PARASITIC ON SUGAR BEETS AND RADISHES. Jour. Agr. Research 4: 279–292, illus.

(21) ELLIS, J. B., and EVERHART, B. M.
1894. NEW SPECIES OF FUNGI FROM VARIOUS LOCALITIES. Acad. Nat. Sci. Phila. Proc. 1894: 322–386.

(22) FITZPATRICK, H. M.
1923. GENERIC CONCEPTS IN THE PYTHIACEAE AND BLASTOCLADIACEAE. Mycologia 15: 166–173.

(23) FROMME, F. D.
1921. THE YELLOWS DISEASE OF CABBAGE IN SOUTHWEST VIRGINIA. Va. Agr. Expt. Sta. Bull. 226, 9 pp., illus.

(24) GARDNER, M. W.
1920. PERONOSPORA IN TURNIP ROOTS. Phytopathology 10: [321]–322.

(25) GILMAN, J. C.
1916. CABBAGE YELLOWS AND THE RELATION OF TEMPERATURE TO ITS OCCURRENCE. Ann. Mo. Bot. Gard. 3: 25–84, illus.

(26) GOLDSWORTHY, M. C.
1926. STUDIES ON THE SPOT DISEASE OF CAULIFLOWER; A USE OF SERUM DIAGNOSIS. Phytopathology 16: 877–884.

(27) HARDING, H. A., and STEWART, F. C.
1904. VITALITY OF PSEUDOMONAS CAMPESTRIS (PAM.) SMITH ON CABBAGE SEED. Science (n. s.) 20: 55–56.

(28) ——— and MORSE, W. J.
1909–10. THE BACTERIAL SOFT ROTS OF CERTAIN VEGETABLES. I: PART 1. THE MUTUAL RELATIONSHIPS OF THE CAUSAL ORGANISMS. N. Y. Agr. Expt. Sta. Tech. Bull. 11: [251]–287, 1909; also in Vt. Agr. Expt. Sta. Bull. 147, pp. [243]–279, 1910.

(29) HEMMI, T.
1923. ON THE RELATION OF TEMPERATURE TO THE DAMPING-OFF OF GARDEN-CRESS SEEDLINGS BY PYTHIUM DEBARYANUM AND CORTICIUM VAGUM. Phytopathology 13: [273]–282, illus.

(30) HENDERSON, M. P.
1918. THE BLACK-LEG DISEASE OF CABBAGE CAUSED BY PHOMA LINGAM (TODE) DESMAZ. Phytopathology 8: [379]–431, illus.

(31) HIGGINS, B. B.
1917. A COLLETOTRICHUM LEAFSPOT OF TURNIPS. Jour. Agr. Research 10: 157–162, illus.

(32) HOLLAND, E. B., and JONES, C. P.
1934. THE RELATION OF "DARK CENTER" TO THE COMPOSITION OF RUTABAGAS. Jour. Agr. Research 48: 377–378.

(33) HUGHES, W.
1933. A STUDY OF PHOMA LINGAM (TODE) DESM. AND OF THE "DRY ROT," ITS CAUSES, PARTICULARLY IN SWEDE TURNIPS. Sci. Proc. Roy. Dublin Soc. (n. s.) 20: 495–529, illus.

(34) JOHNSON, J.
1916. HOST PLANTS OF THIELAVIA BASICOLA. Jour. Agr. Research 7: 289–300, illus.

(35) ——— and HARTMAN, R. E.
1918. INFLUENCE OF SOIL TEMPERATURE ON THIELAVIA ROOT-ROT. (Abstract) Phytopathology 8: 77.

(36) JONES, L. R.
1901. A SOFT ROT OF CARROT AND OTHER VEGETABLES CAUSED BY BACILLUS CAROTOVORUS, JONES. Vt. Agr. Expt. Sta. Rept. (1899–1900) 13: 299–332, illus.

(37) ———
1910. THE BACTERIAL SOFT ROTS OF CERTAIN VEGETABLES. I: PART II, PECTINASE, THE CYTOLYTIC ENZYM PRODUCED BY BACILLUS CAROTOVORUS AND CERTAIN OTHER SOFT-ROT ORGANISMS. N. Y. State Agr. Expt. Sta. Tech. Bull. 11: [289]–368, illus.

(38) ——— WALKER, J. C., and TISDALE, W. B.
1920. FUSARIUM RESISTANT CABBAGE. Wis. Agr. Expt. Sta. Research Bull. 48, 34 pp., illus.

(39) KENDRICK, J. B.
1927. THE BLACK-ROOT DISEASE OF RADISH. Ind. Agr. Expt. Sta. Bull. 311, 32 pp., illus.

(40) ———
1930. KALE YELLOWS IN CALIFORNIA, CAUSED BY FUSARIUM CONGLUTINANS WOLLENW. Hilgardia 5: 1–15, illus.

(41) KUNKEL, L. O.
1918. TISSUE INVASION BY PLASMODIOPHORA BRASSICAE. Jour. Agr. Research 14: 543–572, illus.

(42) ———
1924. FURTHER STUDIES ON THE INTRACELLULAR BODIES ASSOCIATED WITH CERTAIN MOSAIC DISEASES. Hawaii Sugar Planters' Assoc. Expt. Sta. Bull., Bot. Ser. 3: 108–114, illus.

(43) LAURITZEN, J. I.
1929. RHIZOCTONIA ROT OF TURNIPS IN STORAGE. Jour. Agr. Research 38: 93–108, illus.

(44) LARSON, R. H.
1934. WOUND INFECTION AND TISSUE INVASION BY PLASMODIOPHORA BRASSICAE. Jour. Agr. Research 49: 607–624, illus.

(45) ——— and WALKER, J. C.
1934. SOIL TREATMENT IN RELATION TO CLUB-ROOT OF CABBAGE. Jour. Agr. Research 48: 749–759.

(46) LUTMAN, B. F.
1913. STUDIES ON CLUB-ROOT; I. THE RELATION OF PLASMODIOPHORA BRASSICAE TO ITS HOST AND THE STRUCTURE AND GROWTH OF ITS PLASMODIUM. Vt. Agr. Expt. Sta. Bull. 175, 27 pp., illus.

(47) MCCULLOCH, L.
1911. A SPOT-DISEASE OF CAULIFLOWER. U. S. Bur. Plant Indus. Bull. 225, 15 pp., illus.

(48) ———
1929. A BACTERIAL LEAF SPOT OF HORSE-RADISH CAUSED BY BACTERIUM CAMPESTRE VAR. ARMORACIAE, N. VAR. Jour. Agr. Research 38: 269–287, illus.

(49) MACLEOD, D. J., and HOWATT, J. L.
1934. SOIL TREATMENT IN THE CONTROL OF CERTAIN SOIL BORNE DISEASES OF POTATOES. Amer. Potato Jour. 11: 60–61.

(50) MANNS, T. F.
1911. BLACK-LEG OR PHOMA WILT OF CABBAGE. A NEW TROUBLE TO THE UNITED STATES CAUSED BY PHOMA OLERACEA SACC. Phytopathology 1: [28]–31, illus.

(51) MELHUS, I. E.
1911. EXPERIMENTS ON SPORE GERMINATION AND INFECTION IN CERTAIN SPECIES OF OOMYCETES. Wis. Agr. Expt. Sta. Research Bull. 15, pp. [25]–91, illus.

(52) MILBRATH, D. G.
1922. ALTERNARIA FROM CALIFORNIA. Bot. Gaz. 74: 320–324, illus.

(53) MONTEITH, J., JR.
1924. RELATION OF SOIL TEMPERATURE AND SOIL MOISTURE TO INFECTION BY PLASMODIOPHORA BRASSICAE. Jour. Agr. Research 28: 549–562, illus.

(54) Nelson, R.
1926. storage and transportational diseases of vegetables due to suboxidation. Mich. Agr. Expt. Sta. Tech. Bull. 81, 38 pp., illus.
(55) Noffray, E.
1922. le cystope blanc. Jour. Agr. Pratt. (n. s.) 37: 40–41.
(56) Pammel, L. H.
1895. bacteriosis of rutabaga (bacillus campestris n. sp.). Iowa Agr. Expt. Sta. Bull. 27: [130]–134, illus.
(57) Poole, R. F.
1922. the sclerotinia rot of celery. N. J. Agr. Expt. Sta. Bull. 359, 27 pp., illus.
(58) ———
1922. horse-radish root rot investigations. N. J. Agr. Expt. Sta. Repts. (1919–20) 41: 610; (1920–21) 42: 464–465.
(59) ———
1922. bacterial root rot of horseradish in new jersey. (Abstract) Phytopathology 12: 49.
(60) Ramsey, G. B.
1925. sclerotinia species causing decay of vegetables under transit and market conditions. Jour. Agr. Research 31: 597–632, illus.
(61) ———
1935. peronospora in storage cabbage. Phytopathology 25: 955–957, illus.
(62) Reddick, D.
1914. decay of celery in storage. (Abstract) Phytopathology 4: 45.
(63) Rose, D. H., Wright, R. C., and Whiteman, T. M.
1933. the commercial storage of fruits, vegetables, and florists' stocks. U. S. Dept. Agr. Cir. 278, 40 pp.
(64) Schultz, E. S.
1921. a transmissible mosaic disease of chinese cabbage, mustard, and turnip. Jour. Agr. Research 22: 173–178, illus.
(65) Smith, E. F.
1905–11. bacteria in relation to plant disease. v. 1–2. (Carnegie Inst. Pub. 27.)
(66) ———
1920. an introduction to bacterial diseases of plants. Philadelphia and London. 688 pp., illus.
(67) Tisdale, W. B.
1923. influence of soil temperature and soil moisture upon the fusarium disease in cabbage seedlings. Jour. Agr. Research 24: 55–86, illus.
(68) Tompkins, C. M.
1934. a destructive virus disease of cauliflower and other crucifers. (Abstract) Phytopathology 24: 1136–1137.
(69) Walker, J. C.
1922. seed treatment and rainfall in relation to the control of cabbage black-leg. U. S. Dept. Agr. Bull. 1029, 27 pp., illus.
(70) ———
1924. cabbage-seed treatment. U. S. Dept. Agr. Circ. 311, 4 pp., illus.
(71) ———
1927. diseases of cabbage and related plants. U. S. Dept. Agr. Farmers' Bull. 1439, 30 pp., illus.
(72) ———
1933. yellows-resistant lines of jersey wakefield cabbage. Jour. Agr. Research 46: 639–648, illus.
(73) ———
1934. production of cabbage seed free from phoma lingam and bacterium campestre. Phytopathology 24: 158–160.
(74) ———
1936. diseases of vegetable crops. 65 pp. Photo-lithoprint, Edwards Brothers, Inc. Ann Arbor, Mich.
(75) ———, Monteith, J., Jr., and Wellman, F. L.
1927. development of three midseason varieties of cabbage resistant to yellows (fusarium conglutinans woll.). Jour. Agr. Research 35: 785–809, illus.

(76) WALKER, J. C., and SMITH, R.
1930. EFFECT OF ENVIRONMENTAL FACTORS UPON THE RESISTANCE OF CABBAGE TO YELLOWS. Jour. Agr. Research 41: 1–15, illus.
(77) ——— and TISDALE, W. B.
1920. OBSERVATIONS ON SEED TRANSMISSION OF THE CABBAGE BLACK ROT ORGANISM. Phytopathology 10: 175–177.
(78) ——— and WELLMAN, F. L.
1928. A SURVEY OF THE RESISTANCE OF SUBSPECIES OF BRASSICA OLERACEA TO YELLOWS (FUSARIUM CONGLUTINANS). Jour. Agr. Research 37: 233–241, illus.
(79) WEBER, G. F.
1932. SOME DISEASES OF CABBAGE AND OTHER CRUCIFERS IN FLORIDA. Fla. Agr. Expt. Sta. Bull. 256, 62 pp., illus.
(80) WEIMER, J. L.
1925. ALTERNARIA LEAFSPOT AND BROWNROT OF CAULIFLOWER. Jour. Agr. Research 29: 421–441, illus.
(81) ———
1926. RINGSPOT OF CRUCIFERS CAUSED BY MYCOSPHAERELLA BRASSICICOLA (FR.) LINDAU. Jour. Agr. Research 32: 97–132, illus.
(82) ———
1926. A LEAF SPOT OF CRUCIFEROUS PLANTS CAUSED BY ALTERNARIA HERCULEA. Jour. Agr. Research 33: 645–650, illus.
(83) WELLMAN, F. L.
1930. CLUBROOT OF CRUCIFERS. U. S. Dept. Agr. Tech. Bull. 181, 32 pp., illus.
(84) ———
1932. RHIZOCTONIA BOTTOM ROT AND HEAD ROT OF CABBAGE. Jour. Agr. Research 45: 461–469, illus.
(85) WILSON, J. D.
1934. CABBAGE CLUBROOT IN MUCK SOILS. Ohio Agr. Expt. Sta. Bimonthly Bull. 167: 58–65, illus.
(86) WOLF, F. A.
1918. INTUMESCENCES, WITH A NOTE ON MECHANICAL INJURY AS A CAUSE OF THEIR DEVELOPMENT. Jour. Agr. Research 13: 253–260, illus.
(87) WRIGHT, R. C., and TAYLOR, G. F.
1923. THE FREEZING TEMPERATURES OF SOME FRUITS, VEGETABLES, AND CUT FLOWERS. U. S. Dept. Agr. Bull. 1133, 8 pp.

CUCURBITS

(88) AINSWORTH, G. C.
1935. VIRUS DISEASES OF CUCUMBER. [Gt. Brit.] Jour. Min. Agr. 42: 338–344, illus.
(89) ARTHUR, J. C.
1889. SPOTTING OF PEACHES AND CUCUMBERS. Ind. Agr. Expt. Sta. Bull. 19, 10 pp., illus.
(90) BAILEY, R. M., and BURGESS, I. M.
1935. BREEDING CUCUMBERS RESISTANT TO SCAB. Amer. Soc. Hort. Sci. Proc. (1934) 32: 474–476.
(91) BOYD, O. C.
1926. FURTHER EXPERIMENTS IN THE CONTROL OF WATERMELON ANTHRACNOSE WITH DUSTS AND SPRAYS. (Abstract) Phytopathology 16: 641, 642.
(92) BRISLEY, H. R.
1923. STUDIES ON THE BLIGHT OF CUCURBITS CAUSED BY MACROSPORIUM CUCUMERINUM E. & E. Phytopathology 13: [199]–204, illus.
(93) BROWN, J. G., and EVANS, M. M.
1933. A PHYTOPHTHORA ROT OF WATERMELON. Ariz. Agr. Expt. Sta. Tech. Bull. 51, pp. [45]–65, illus.
(94) BRYAN, M. K.
1930. BACTERIAL LEAF SPOT OF SQUASH. Jour. Agr. Research 40: 385–391, illus.
(95) BURGER, O. F.
1913. A BACTERIAL ROT OF CUCUMBERS. Phytopathology 3: 169–170.
(96) ———
1914. CUCUMBER ROT. Fla. Agr. Expt. Sta. Bull. 121, pp. [97]–109, illus.

(97) CARON, O.
1928. THE CUCUMBER GALL. Quebec Soc. Protect. Plants Ann. Rept. (1927–28) 20: 69–70.

(98) CARSNER, E.
1918. ANGULAR-LEAFSPOT OF CUCUMBER: DISSEMINATION, OVERWINTERING, AND CONTROL. Jour. Agr. Research 15: 201–220, illus.

(99) CHUPP, C.
1925. MANUAL OF VEGETABLE-GARDEN DISEASES. 647 pp., illus. New York.

(100) CHURCH, M. B.
1921. THE RELATION OF MOSAIC DISEASE TO PICKLING OF CUCUMBERS. (Phytopath. Note) Phytopathology 11: 28–29.

(101) CLAYTON, E. E.
1927. EFFECT OF EARLY SPRAY AND DUST APPLICATIONS ON LATER INCIDENCE OF CUCUMBER WILT AND MOSAIC DISEASES. Phytopathology 17: 473–481.

(102) ———.
1929. BREEDING FOR RESISTANCE TO CUCUMBER MOSAIC DISEASE. (Abstract) Phytopathology 19: 85.

(103) ———.
1931. CUCUMBER DISEASE INVESTIGATIONS ON LONG ISLAND. N. Y. State Agr. Expt. Sta. Bull. 590, 20 pp., illus.

(104) CLINTON, G. P.
1905. DOWNY MILDEW, OR BLIGHT, PERONOPLASMOPARA CUBENSIS (B. & C.) CLINT., OF MUSKMELONS AND CUCUMBERS. Conn. Agr. Expt. Sta. Ann. Rept. (1904) 28: 329–362, illus.

(105) CURRENCE, T. M., and LEACH, J. G.
1935. PROGRESS IN DEVELOPING MUSKMELON STRAINS RESISTANT TO FUSARIUM. Amer. Soc. Hort. Sci. Proc. (1934) 32: 481–482.

(106) DAWSON, M. L.
1931. A STUDY OF SOME VEGETABLE ROTS OCCURRING IN EVANSTON MARKETS. Ill. State Acad. Sci. Trans. (1930) 23: 131–142, illus.

(107) DOOLITTLE, S. P.
1916. CUCUMBER SCAB CAUSED BY CLADOSPORIUM CUCUMERINUM. Mich. Acad. Sci. Ann. Rept. 17: [87]–116, illus.

(108) ———
1916. A NEW INFECTIOUS MOSAIC DISEASE OF CUCUMBER. Phytopathology 6: [145]–147.

(109) ———
1920. THE MOSAIC DISEASE OF CUCURBITS. U. S. Dept. Agr. Bull. 879, 69 pp., illus.

(110) ———
1921. OVERWINTERING OF THE BACTERIAL WILT OF CUCURBITS. (Phytopath. Note) Phytopathology 11: 299–300.

(111) ———
1922. COMPARATIVE SUSCEPTIBILITY OF EUROPEAN AND AMERICAN VARIETIES OF CUCUMBERS TO BACTERIAL WILT. Phytopathology 12: [143]–146.

(112) ———
1924. CONTROL OF CUCUMBER MOSAIC IN THE GREENHOUSE. U. S. Dept. Agr. Cir. 321, 6 pp., illus.

(113) ——— and GILBERT, W. W.
1919. SEED TRANSMISSION OF CUCURBIT MOSAIC BY THE WILD CUCUMBER. Phytopathology 9: [326]–327.

(114) ——— and WALKER, M. N.
1925. FURTHER STUDIES ON THE OVERWINTERING AND DISSEMINATION OF CUCURBIT MOSAIC. Jour. Agr. Research 31: 1–58, illus.

(115) ——— and WALKER, M. N.
1926. CONTROL OF CUCUMBER MOSAIC BY ERADICATION OF WILD HOST PLANTS. U. S. Dept. Agr. Bull. 1461, 15 pp., illus.

(116) DORAN, W. L.
1932. DOWNY MILDEW OF CUCUMBERS. Mass. Agr. Expt. Sta. Bull. 283, 22 pp.

(117) DRECHSLER, C.
1923. A NEW BLOSSOM-END DECAY OF WATERMELONS CAUSED BY AN UNDESCRIBED SPECIES OF PYTHIUM. (Abstract) Phytopathology 13: 57.
(118) ———
1925. THE COTTONY LEAK OF CUCUMBERS CAUSED BY PYTHIUM APHANIDERMATUM. Jour. Agr. Research 30: 1035–1042, illus.
(119) ———
1929. A FRUIT ROT OF HONEY DEW MELONS DUE TO A SPECIES OF PHYTOPHTHORA. (Abstract) Phytopathology 19: 85.
(120) ———
1930. SOME NEW SPECIES OF PYTHIUM. Jour. Wash. Acad. Sci. 20: 398–418.
(121) EDGERTON, C. W.
1915. EFFECT OF TEMPERATURE ON GLOMERELLA. Phytopathology 5: [247]–259, illus.
(122) ELLIOTT, J. A.
1917. TAXONOMIC CHARACTERS OF THE GENERA ALTERNARIA AND MACROSPORIUM. Amer. Jour. Bot. 4: 439–476, illus.
(123) FISCHER, G. W.
1930. A STUDY OF THE FRUIT DISEASES OCCURRING IN A MID-WESTERN MARKET. Butler Univ. Bot. Studies 1 (Paper 8): 105–127, illus.
(124) FULTON, H. R., and WINSTON, J. R.
1915. WATERMELON WILT SPREAD BY CONTAMINATED SEED. N. C. Agr. Expt. Sta. Bien. Rpt. (1913–14) 36: 48–51, illus.
(125) GARDNER, M. W.
1918. ANTHRACNOSE OF CUCURBITS. U. S. Dept. Agr. Bull. 727, 68 pp., illus.
(126) ——— and GILBERT, W. W.
1921. FIELD TESTS WITH CUCUMBER ANGULAR LEAF-SPOT AND ANTHRACNOSE. (Phytopath. Note) Phytopathology 11: 298–299.
(127) GIDDINGS, N. J.
1910. A BACTERIAL SOFT ROT OF MUSKMELON, CAUSED BY BACILLUS MELONIS N. SP. Vt. Agr. Expt. Sta. Bull. 148, pp. 366–416, illus.
(128) GILBERT, W. W.
1916. CUCUMBER MOSAIC DISEASE. Phytopathology 6: [143]–144, illus.
(129) ———
1917. VIRULENCE OF DIFFERENT STRAINS OF CLADOSPORIUM CUCUMERINUM. (Abstract) Phytopathology 7: 62.
(130) ——— and GARDNER, M. W.
1918. SEED TREATMENT CONTROL AND OVERWINTERING OF CUCUMBER ANGULAR LEAF-SPOT. Phytopathology 8: [229]–233, illus.
(131) ——— and MEIER, F. C.
1926. CHEMICAL INJURY TO WATERMELONS IN TRANSIT. (Abstract) Phytopathology 16: 73.
(132) ——— and MEIER, F. C.
1929. CHEMICAL INJURY TO WATERMELONS IN TRANSIT. U. S. Dept. Agr. Cir. 74, 10 pp., illus.
(133) ——— and MEIER, F. C.
1929. CHEMICAL INJURY TO WATERMELONS IN TRANSIT—RESULTS OF SHIPPING TESTS. (Abstract) Phytopathology 19: 85.
(134) GRIFFIN, H. H.
1901. THE CANTALOUPE. Colo. Agr. Expt. Sta. Bull. 62, 18 pp., illus.
(135) GROSSENBACHER, J. G.
1909. A MYCOSPHAERELLA WILT OF MELONS. N. Y. Agr. Expt. Sta. Tech. Bull. 9, pp. [195]–229, illus.
(136) GUBA, E. F.
1928. CONTROL OF CUCUMBER POWDERY MILDEW IN GREENHOUSES. Phytopathology 18: 847–860, illus.
(137) HARTER, L. L., and WEIMER, J. L.
1922. DECAY OF VARIOUS VEGETABLES AND FRUITS BY DIFFERENT SPECIES OF RHIZOPUS. Phytopathology 12: [205]–212.
(138) HEMMI, T.
1922. ON THE OCCURRENCE OF MYCOSPHAERELLA WILT OF MUSKMELONS IN JAPAN. Phytopathology 12: [394]–397.

(139) HOGGAN, I. A.
1930. TRANSMISSION OF CUCUMBER MOSAIC TO SPINACH. Phytopathology 20: 103–105, illus.
(140) ———
1935. TWO VIRUSES OF THE CUCUMBER MOSAIC GROUP ON TOBACCO. Ann. Appl. Biol. 22: 27–36, illus.
(141) HUMPHREY, J. E.
1893. THE LEAF BLIGHT—CLADOSPORIUM CUCUMERINUM ELL. & ARTH. Mass. State Agr. Expt. Sta. Ann. Rept. (1892) 10: 227–229.
(142) JAGGER, I. C.
1916. EXPERIMENTS WITH THE CUCUMBER MOSAIC DISEASE. Phytopathology 6: [148]–151.
(143) ———
1917. TWO TRANSMISSIBLE MOSAIC DISEASES OF CUCUMBERS. (Abstract) Phytopathology 7: 61.
(144) ———
1918. HOSTS OF THE WHITE PICKLE MOSAIC DISEASE OF CUCUMBER. (Phytopath. Note) Phytopathology 8: 32–33.
(145) ———
1926. POWDERY MILDEW OF MUSKMELONS IN THE IMPERIAL VALLEY OF CALIFORNIA IN 1925. (Phytopath. Note) Phytopathology 16: 1009–1010.
(146) ——— and SCOTT, G. W.
1931. MELONS RESISTANT TO POWDERY MILDEW. (Abstract) Phytopathology 21: 113–114.
(147) ——— and SCOTT, G. W.
1937. DEVELOPMENT OF POWDERY MILDEW RESISTANT CANTALOUP NO. 45. U. S. Dept. Agr. Cir. 441, 6 pp., illus.
(148) JEHLE, R. A., and POTTS, S. F.
1924. DUSTING AND SPRAYING CANTALOUPES. Md. Agr. Expt. Sta. Bull. 263, pp. [169]–180, illus.
(149) JONES, L. R., and DOOLITTLE, S. P.
1921. ANGULAR LEAF-SPOT OF CUCUMBER. (Phytopath. Note) Phytopathology 11: [297]–298.
(150) KAUFMAN, C. H.
1921. A BLACK ROT OF SQUASH. Mich. Acad. Sci., Arts and Letters Ann. Rept. 22: 201–202.
(151) KENDRICK, J. B.
1934. CUCURBIT MOSAIC TRANSMITTED BY MUSKMELON SEED. Phytopathology 24: 820–823, illus.
(152) LAYTON, D. V.
1932. HOST RESPONSE OF CITRULLUS VULGARIS TO COLLETOTRICHUM LAGENARIUM. (Abstract) Phytopathology 22: 16.
(153) ——— and WILSON, J. J.
1931. THREE NEW WILT-RESISTANT VARIETIES OF WATERMELON. (Abstract) Phytopathology 21: 114.
(154) LEACH, J. G.
1933. A DESTRUCTIVE FUSARIUM WILT OF MUSKMELONS. (Phytopath. Note) Phytopathology 23: 554–556, illus.
(155) ——— and CURRENCE, T. M.
1935. RESISTANCE TO FUSARIUM WILT IN MUSKMELONS. (Abstract) Phytopathology 25: 25.
(156) LECLERG, E. L.
1931. THE RELATION OF LEAF BLIGHT TO SUN SCALD OF HONEYDEW MELONS. (Phytopath. Note) Phytopathology 21: 97–98, illus.
(157) LEWIS, C. E.
1913. COMPARATIVE STUDIES OF CERTAIN DISEASE PRODUCING SPECIES OF FUSARIUM. Maine Agr. Expt. Sta. Bull. 219, pp. [203]–258, illus.
(158) LINK, G. K. K., and GARDNER, M. W.
1919. MARKET PATHOLOGY AND MARKET DISEASES OF VEGETABLES. Phytopathology 9: [497]–520.
(159) ——— and MEIER, F. C.
1922. ANTHRACNOSE OF MUSKMELONS. U. S. Dept. Agr. Cir. 217, 4 pp., illus.

(160) MAHONEY, C. H.
1935. SEED TRANSMISSION OF MOSAIC IN INBRED LINES OF MUSKMELONS (CUCUMIS MELO L.). Amer. Soc. Hort. Sci. Proc. (1934) 32: 477–480.
(161) MARTIN, W. H.
1917. COMMON DISEASES OF CUCUMBERS AND MELONS. N. J. Agr. Expt. Sta. Cir. 68, 12 pp., illus.
(162) MEIER, F. C.
1916. WATERMELON STEM-END ROT [PRELIMINARY PAPER]. Jour. Agr. Research 6: 149–152, illus.
(163) ———
1920. CONTROL OF WATERMELON ANTHRACNOSE BY SPRAYING. U. S. Dept. Agr. Cir. 90, 11 p., illus.
(164) ———, DRECHSLER, C., and EDDY, E.
1922. CUCUMBER BLACK ROT CAUSED BY MYCOSPHAERELLA CITRULLINA. (Abstract) Phytopathology 12: 43.
(165) ——— and LINK, G. K. K.
1922. BACTERIAL SPOT OF CUCUMBERS. U. S. Dept. Agr. Cir. 234, 5 pp., illus.
(166) MELHUS, I. E., and ELMER, O. H.
1925. DISEASES OF CUCUMBERS AND MELONS IN IOWA. Iowa Agr. Expt. Sta. Cir. 99, 16 pp., illus.
(167) MILLER, P. A., and BARRETT, J. T.
1931. CANTALOUPE POWDERY MILDEW IN THE IMPERIAL VALLEY. Calif. Agr. Expt. Sta. Bull. 507, 36 pp., illus.
(168) MUYXENBERG, E. W. B. V. D.
1932. ONDERZOEK OVER CLADOSPORIUM CUCUMERINUM ELLIS & ARTHUR (DE VEROORZAKER VAN HET VRUCHTVUUR VAN DE KOMKOMMER). Tijdschr. Plantenziekten 38: [81]–118, illus. [In Dutch. English summary, pp. 114–115.]
(169) ORTON, W. A.
1905. SPRAYING FOR CUCUMBER AND MELON DISEASES. U. S. Dept. Agr. Farmers' Bull. 231, 24 pp., illus.
(170) ———
1913. THE DEVELOPMENT OF DISEASE RESISTANT VARIETIES OF PLANTS. 4th Conf. Internat. Genetique, Paris, 1911, Compt. Rend. et Rap., pp. 247–265, illus.
(171) ——— and MEIER, F. C.
1922. DISEASES OF WATERMELONS. U. S. Dept. Agr. Farmers' Bull. 1277, 31 pp., illus.
(172) PFÄLTZER, A. C. B.
1927. HET VRUCHT– EN BLADVUUR VAN DE KOMKOMMER. CLADOSPORIUM CUCUMERINUM, ELL. ET ARTH. EN CORYNESPORA MELONIS, (COOKE), LINDAU. 73 pp., illus. Baarn (Thesis, Univ. Utrecht).
(173) PORTER, D. R.
1928. INFECTION STUDIES WITH WATERMELON WILT CAUSED BY FUSARIUM NIVEUM EFS. Iowa Agr. Expt. Sta. Research Bull. 112, pp. [346]–368, illus.
(174) ———
1932. SOME ENVIRONMENTAL RELATIONS OF WATERMELON WILT. Phytopath. 22: 813–825, illus.
(175) ——— and MELHUS, I. E.
1932. THE PATHOGENICITY OF FUSARIUM NIVEUM (EFS.) AND THE DEVELOPMENT OF WILT RESISTANT STRAINS OF CITRULLUS VULGARIS (SCHRAD.). Iowa Agr. Expt. Sta. Research Bull. 149, pp. [123]–184a, illus.
(176) PORTER, R. H.
1930. A NEW MOSAIC DISEASE OF CUCUMBER. (Abstract) Phytopathology 20: 113.
(177) ———
1931. THE REACTION OF CUCUMBERS TO TYPES OF MOSAIC. Iowa State Col. Jour. Sci. 6: 95–129, illus.
(178) PRICE, W. C.
1934. ISOLATION AND STUDY OF SOME YELLOW STRAINS OF CUCUMBER MOSAIC. Phytopathology 24: 743–761, illus.

(179) RAND, F. V.
1915. DISSEMINATION OF BACTERIAL WILT OF CUCURBITS. [Preliminary note.] Jour. Agr. Research 5: 257–260, illus.

(180) ——— and CASH, L. C.
1920. SOME INSECT RELATIONS OF BACILLUS TRACHEIPHILUS ERW. SM. Phytopathology 10: [133]–140, illus.

(181) ——— and ENLOWS, E. M. A.
1916. TRANSMISSION AND CONTROL OF BACTERIAL WILT OF CUCURBITS. Jour. Agr. Research 6: 417–434, illus.

(182) ——— and ENLOWS, E. M. A.
1920. BACTERIAL WILT OF CUCURBITS. U. S. Dept. Agr. Bull. 826, 43 pp., illus.

(183) SHELDON, J. L.
1904. DISEASES OF MELONS AND CUCUMBERS DURING 1903 AND 1904. W. Va. Agr. Expt. Sta. Bull. 94, pp. [121]–138, illus.

(184) SLEETH, B.
1934. FUSARIUM NIVEUM, THE CAUSE OF WATERMELON WILT. W. Va. Agr. Expt. Sta. Bull. 257, 23 pp., illus.

(185) SMITH, E. F.
1899. WILT DISEASE OF COTTON, WATERMELON, AND COWPEA (NEOCOSMOSPORA NOV. GEN.). U. S. Dept. Agr., Div. Veg. Physiol. and Path. Bull. 17, 72 pp., illus.

(186) ——— and BRYAN, M. K.
1915. ANGULAR LEAF-SPOT OF CUCUMBERS. Jour. Agr. Research 5: 465–476, illus.

(187) STEVENS, F. L.
1931. THE ASCIGEROUS STAGE OF COLLETOTRICHUM LAGENARIUM INDUCED BY ULTRA-VIOLET IRRADIATION. Mycologia 23: 134–139, illus.

(188) STEWART, F. C.
1897. THE DOWNY MILDEW OF THE CUCUMBER; WHAT IT IS AND HOW TO PREVENT IT. N. Y. State Agr. Expt. Sta. Bull. 119, pp. 155–182, illus.

(189) STURGIS, W. C.
1896. A DISEASE OF MELONS (ALTERNARIA SP.). Conn. State Agr. Expt. Sta. Ann. Rept. (1895) 19: 186–187.

(190) TAUBENHAUS, J. J.
1920. WILTS OF THE WATERMELON AND RELATED CROPS (FUSARIUM WILTS OF CUCURBITS). Tex. Agr. Expt. Sta. Bull. 260, 50 pp., illus.

(191) ——— and EZEKIEL, W. N.
1933. A NEW CHARCOAL ROT OF CANTALOUPES. Tex. Agr. Expt. Sta. Ann. Rept. (1932) 45: 74–75.

(192) ——— and EZEKIEL, W. N.
1933. A NEW GRAY ROT OF CANTALOUPES. Tex. Agr. Expt. Sta. Ann. Rept. (1932) 45: 74.

(193) ——— and EZEKIEL, W. N.
1934. A NEW GRAY ROT OF CUCUMBERS. Tex. Agr. Expt. Ann. Rept. (1933) 46: 88.

(194) ——— and EZEKIEL, W. N.
1934. STEM-END ROT OF WATERMELONS. Tex. Agr. Expt. Sta. Ann. Rept. (1933) 46: 88.

(195) TEMPLE, C. E.
1931. SOME DISEASES OF CANTALOUPE AND CUCUMBER. Peninsula Hort. Soc. [Del.] Trans. 45: 35–41.

(196) VAN HALTERN, F.
1930. SEED TREATMENT FOR THE CONTROL OF SEED-BORNE DISEASES OF MELON, CUCUMBER, AND SQUASH. Ga. Expt. Sta. Cir. 88, 8 pp.

(197) ———
1933. SPRAYING CANTALOUPES FOR THE CONTROL OF DOWNY MILDEW AND OTHER DISEASES. Ga. Agr. Expt. Sta. Bull. 175, 53 pp., illus.

(198) WALKER, M. N.
1925. THE RELATION OF CERTAIN SPECIES OF PHYSALIS TO THE OVERWINTERING OF THE MOSAIC DISEASE OF CUCUMBER. Phytopathology 15: [733]–744, illus.

(199) ———
1926. A COMPARATIVE STUDY OF THE MOSAIC DISEASES OF CUCUMBER, TOMATO, AND PHYSALIS. Phytopathology 16: 431–458.

(200) ———
1933. OCCURRENCE OF WATERMELON MOSAIC. Phytopathology 23: 741–744, illus.

(201) ——— and WEBER, G. F.
1931. DISEASES OF WATERMELONS IN FLORIDA. Fla. Agr. Expt. Sta. Bull. 225, 52 pp., illus.

(202) WEBER, G. F.
1929. ANGULAR LEAF SPOT AND FRUIT ROT OF CUCUMBERS CAUSED BY BACTERIUM LACHRYMANS E. F. S. & BRY. Fla. Agr. Expt. Sta. Bull. 207, 32 pp., illus.

(203) ———
1929. CUCUMBER DISEASES IN FLORIDA. Fla. Agr. Expt. Sta. Bull. 208, 48 pp., illus.

(204) ———
1931. SPRAYING AND DUSTING CUCUMBERS FOR CONTROL OF DOWNY MILDEW FROM 1925 TO 1930. Fla. Agr. Expt. Sta. Bull. 230, 58 pp., illus.

(205) WEIMER, J. L., and HARTER, L. L.
1923. TEMPERATURE RELATIONS OF ELEVEN SPECIES OF RHIZOPUS. Jour. Agr. Research 24: 1–40, illus.

(206) WIANT, J. S.
1937. INVESTIGATIONS OF THE MARKET DISEASES OF CANTALOUPS AND HONEY DEW AND HONEY BALL MELONS. U. S. Dept. Agr. Tech. Bull. 573, 48 pp., illus.

(207) WOLF, F. A.
1917. A SQUASH DISEASE CAUSED BY CHOANEPHORA CUCURBITARUM. Jour. Agr. Research 8: 319–328, illus.

(208) ZIMMERLEY, H. H., DAVIS, R. J., and SPENCER, H.
1923. SPRAYING AND DUSTING CANTALOUPES. Va. (Truck Sta.) Agr. Expt. Sta. Bull. 45, pp. [295]–313, illus.

U. S. GOVERNMENT PRINTING OFFICE: 1938